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EDITORIAL · REDAKSIONEEL

1960: A YEAR OF DECISION

Recent developments in schemes for pre-paid medical care have focused attention on the coming year as one which may well determine the continued existence of the medical profession as we have for long known it. In the Transvaal particularly, the area of private practice has increasingly been encroached upon by contract practice of various kinds, until to-day it is fair to say that private patients have dwindled to some 10–20% of what the position was some 15–20 years ago.

There is no harm in the progressive development of contract practice, provided the standards of medical care are not lowered and the profession is not placed in economic jeopardy. Furthermore, one of the dangers which must be watched very closely is the possible development of a monopoly of consumers who may find themselves in a position to dictate to the profession the conditions under which medical practitioners shall work, live and earn. There is a real danger that the profession may be deprived of the right to control its own destiny, unless it takes timely steps to secure its independence and autonomy.

We welcome any attempt to spread the economic risks of illness and we endorse any form of insurance which will alleviate the financial burden (in whole or in part) of hospital and medical care. But the establishment of lay-controlled organizations (constructed on a profit basis—whether the profit derives directly or obliquely from health insurance activities) will

1960: 'N JAAR VAN BESLISSING

Onlangse ontwikkelinge in die skemas i.v.m. vooruitbetaalde mediese behandeling het die aandag op die komende jaar gevestig as die tydvak wat bes moontlik 'n beslissing kan vel oor die voortbestaan van die mediese professie soos ons dit reeds so lank ken. Veral in Transvaal het kontrakpraktyk van verskillende soorte in so 'n mate inbreuk op die gebied van private praktyk gedoen dat daar vandag met 'n baie groot mate van waarheid gesê kan word dat private pasiënte tot ongeveer 10–20% gedaal het in vergelyking met wat die posisie naastenby 15 of 20 jaar gelede was.

Daar is geen kwaad in die progressiewe ontwikkeling van kontrakpraktyk nie, mits die maatstawwe van mediese versorging nie verlaag en die professie nie in ekonomiese gevaar gestel word nie. Temeer, een van die gevare wat baie sorgvuldig dopgehou moet word, is die moontlike ontwikkeling van 'n monopolie van verbruikers wat later in so 'n sterk posisie kan verkeer dat hulle die voorwaardes waaronder geneeshere werk, lewe en hul geld verdien, sal kan voorskryf. Daar is 'n wesenlike gevaar dat die professie beroof kan word van die reg om oor sy eie lotgevalle te beslis, tensy stappe betyds gedoen word om die onafhanklikheid en outonomie van geneeshere te verseker.

Ons verwelkom enige poging om die ekonomiese risiko's van 'n siekte te versprei, en ons heg ons goedkeuring aan alle vorms van versekering wat die finansiële las van hospitaal- en mediese behandeling geheel en al of gedeeltelik sal verlig. Maar die stigting van leke-

lead to economic competition between all the organizations concerned to provide the insurance cover at the cheapest rate. Unilateral action on the part of powerful and influential bodies may well result in the cheapest (or cheaper) cover being provided at the expense of the profession; and in any negotiations with the organized profession there must be concern and care to preserve the existing pattern of private practice and professional autonomy.

Lay-controlled commercial organizations competing among themselves for the sale of sickness insurance at the lowest possible premiums must not be confused with Friendly Societies organized on a mutual non-profit basis, whether these are controlled by the members themselves (as in the case of Medical Aid Societies) or by the profession (as in the case of the Medical Association-sponsored Medical Services Plan).

The profession should oppose strongly any suggestion that commercial insurance companies should enjoy the same or similar privileges accorded by practitioners as an act of charity to Medical Aid Societies. There is no basis on which these two wholly dissimilar types of organization can be equated; and the profession will be well advised not to take any action which may harm either existing Medical Aid Societies (which have for so many years co-operated so loyally with the Medical Association) or the Medical Services Plan (the scheme officially sponsored by the Medical Association itself).

The coming months are fraught with difficulties for medical practitioners in private practice. Imagination and foresight are needed if we are not to witness in the course of this year the first stages of the disintegration of the unity and security of the profession.

organisasies (opgebou op 'n winsbasis—of daardie wins noual regstreeks of onregstreeks uit gesondheidsversekeringsbedrywighede voortspruit) sal aanleiding gee tot ekonomiese mededinging tussen al die organisasies wat hulle daarop toelê om versekeringsdekking teen die goedkoopste premietarief te verskaf. Eensydige optrede aan die kant van kragtige en invloedryke liggame kan bes moontlik tot gevolg hê dat die goedkoopste (of goedkoper) dekking verskaf word ten koste van die professie; en in enige onderhandelinge met die georganiseerde professie moet daar aandag en sorg bestee word aan die handhawing van die huidige patroon van private praktyk en professionele outonomie.

Handelsorganisasies wat deur leke gekontroleer word en met mekaar meeding om die verkoop van siekteversekering teen die laags moontlike premie moet nie verwar word met onderstandsverenigings wat op 'n onderlinge nie-winssoekende grondslag georganiseer is nie—of laasgenoemde nou al gekontroleer word deur die lede self (soos in die geval van mediese hulpverenigings) of deur die professie (soos in die geval van die Mediese Dienste-plan wat onder beskerming van die Mediese Vereniging staan).

Die professie behoort sterk te velde te trek teen enige suggestie dat kommersiële versekeringsmaatskappye dieselfde of dergelike voorregte moet geniet as die mediese onderstandsverenigings waaraan mediese praktisyns, as 'n liefdedaad, sekere voorregte toegestaan het. Daar is geen grondslag waarop hierdie twee volkome verskillende soorte organisasies gelykgestel kan word nie; en die professie sou verstandig handel as hy geen stappe doen nie wat bes moontlik skade kan berokken aan die huidige mediese onderstandsverenigings (wat baie jare lank heelhartig met die Mediese Vereniging saamgewerk het) of die Mediese Dienste-plan, d.w.s. die skema wat amptelik deur die Mediese Vereniging self van stapel gestuur is.

Die komende maande sal talle moeilikhede vir private mediese praktisyns meebring. As verbeelding en oorleg nie aan die dag gelê word nie, is dit moontlik dat ons gedurende die eersvolgende twaalf maande die eerste stadiums van die desintegrasie van die eenheid en veiligheid van die professie sal aanskou.

OUR BARAGWANATH NUMBER

In May 1958 we were privileged to publish in *Medical Proceedings* a special Baragwanath Hospital Number to commemorate the decennial anniversary of that hospital.

It is therefore all the more gratifying to be able to acquaint our colleagues about further work which is reported from Baragwanath Hospital in this as well as in the next issue of *Medical Proceedings*.

Baragwanath Hospital with its 2,000 beds, is the largest hospital not only in the Commonwealth but also on the Continent of Africa. Its plans for extension include an

ONS BARAGWANATH-NOMMER

In Mei 1958 was ons bevoorreg om 'n spesiale Baragwanath-hospitaalnommer in *Mediese Bydraes* te publiseer toe die hospitaal sy tienjarige bestaan gevier het. Dit is derhalwe des te meer verblydend dat ons in hierdie sowel as in die volgende uitgawe van *Mediese Bydraes* ons kollegas op hoogte kan bring van die verdere werk wat sedertdien by die Baragwanath-hospitaal gedoen is.

Die Baragwanath-hospitaal met sy 2,000 beddens is die grootste hospitaal nie alleen in die Statebond nie, maar ook op die vasteland van Afrika. Die uitbreidingsplanne maak

orthopaedic hospital (116 beds) and additional maternity facilities involving a further 375 beds. It already provides facilities for post-graduate clinical work and is undertaking post-graduate training of orthopaedic and paediatric nurses, as well as sister-tutor courses.

The hospital plays an important rôle in research in Southern Africa. Its resources are particularly appropriate for one of its most recent undertakings, viz. participation in an extensive research project on the etiology of coronary artery disease. This programme has been made possible by a grant of £50,000 from the U.S. National Health Services.

There seems little doubt that Baragwanath Hospital will become one of the world's important centres for treatment, training and research.

voorsiening vir 'n ortopediese hospitaal (116 beddens) en addisionele kraamverplegingsfasiliteite waarvoor 'n verdere 275 beddens nodig sal wees. Dit verskaf reeds fasiliteite vir na-graadse kliniese opleiding, en die na-graadse opleiding van ortopediese en pediatriese verpleegsters, sowel as kursusse vir susterdosente, sal eersdaags onderneem word.

Die hospitaal speel 'n belangrike rol vir sover dit navorsing in Suidelike Afrika betref. Sy hulpbronne is by uitstek geskik vir een van sy jongste ondernemings, naamlik deelneming aan 'n uitgebreide navorsingskema na die etiologie van koronêre hartkwaal. Hierdie program is moontlik gemaak deur 'n toelae van £50,000 wat van die U.S. National Health Services ontvang is.

Dit skyn byna seker te wees dat Baragwanath-hospitaal een van die wêreld se belangrike sentrums vir behandeling, opleiding en navorsing gaan word.

THE PHYSICIAN'S PRAYER

(Based on a prayer in prose by Anon)

Dear Lord, grant this a doctor's humble prayer:
'Help me neither to falter nor evade,
Cleanse me from prejudice and stubborn pride,
For these breed false beliefs and egotism.
Let me assume no skills, but justly earned,
And mark for me, Integrity, a precious gift.
Let me cleave to Truth and not adorn
My mind with fatuous thoughts and jealousies.
Guard me 'gainst evil—e'en in subtlest form,
Yet help me tolerate the faults of others.
Guide my hand too in those despairing hours
When I have erred in tasks beyond my skill
And yet, let Courage ever be my garment.
Deliver me from crude and callous acts,
Thus keep me human—withal free from maudlin traits,
That mine—the Physician's mind—be bright as steel.
'Bove all, teach me the priceless Joy of healing,
That when I stand on trial at Judgment Day
I may be tranquil in the sure belief
That I helped soothe Life's restless tragedy'

. . . . *Amen.*

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TETANUS NEONATORUM

A REVIEW OF 53 CASES

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Tetanus neonatorum has a high mortality rate which varies from 71% to 96% in reports dealing with large numbers of cases.[1-7] Experimental and clinical proof of the anti-tetanus effect of chlorpromazine have raised hopes of lowering the at present formidable mortality rate. In 1957, when reports began to appear in the literature of the use of chlorpromazine in the treatment of tetanus, we decided to try this drug in combination with promethazine and pethidine.[8-11]

Since then 25 patients have been treated with this intravenous chlorpromazine 'cocktail.' The results of this therapy are analysed in this paper and they are compared with those obtained in this Department before April 1957, when our treatment consisted chiefly of nasogastric feeding and phenobarbitone as the anticonvulsant.

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Since factors other than the treatment influence the prognosis of tetanus neonatorum, the 53 patients in this series are described in some detail.

None of the infants was born in hospital. Most had been delivered by a relative or friend. Usually the infant had been well for a few days after delivery and then began to refuse feeds or was unable to suck. Some of the mothers observed that this was due to stiffness of the jaws. Shortly thereafter generalized body stiffness was noted with the onset of spasms. A few babies were brought to hospital because they turned blue during an attack. The characteristic clinical signs were trismus, risus sardonicus, widespread muscular rigidity and reflex spasms. In a few cases diagnosis presented difficulties and lumbar punctures were carried out. Examination of the cerebrospinal fluid proved to be normal in all patients referred to in this paper.

METHODS

The weight of each infant was recorded on admission. The incubation period was equated to the number of days from birth to onset of symptoms. The duration of symptoms before admission was also noted.

The patients were divided into 2 groups:

Group A, consisting of 28 children were admitted to hospital before April 1957 and who were treated with phenobarbitone and other sedatives; and

Group B, consisting of 25 patients admitted after May 1957 who were treated with the intravenous 'cocktail' of chlorpromazine, promethazine and pethidine.

The infant was accommodated in a quiet room, placed in a 'Queen Charlotte' tent and given oxygen at 2–4 litres per minute. A special nurse attended to the infant. It was her duty to regulate the rate of the intravenous drip, to turn the baby and record the pulse, respiration and rectal temperature every 2 hours and to notify the doctor if there were more than 6 spontaneous spasms per hour. In both groups the umbilicus was cleaned with hydrogen peroxide shortly after admission. Anti-tetanus serum was given, half by intramuscular and half by intravenous injection, in average doses of 72,000 units before April 1957 and 45,000 units after this date.

Patients in Group A were fed via a naso-gastric tube every 3 hours usually with expressed breast milk. The sedative was phenobarbitone, in doses of 1 grain every 8 hours via the naso-gastric polythene tube. Where this did not control spasms, paraldehyde was given additionally in doses of 1 c.c. by intramuscular injection every 2–6 hours. In 4 instances rectal bromethol (Avertin) was used as an additional sedative. Penicillin was administered in doses of 100,000 to 250,000 units by intramuscular injection every 6–8 hours.

All infants in Group B received 1 c.c. of paraldehyde by intramuscular injection before a cut-down on the saphenous vein was performed. Then an injection was given via the drip tubing of a mixture of chlorpromazine (1.0–2.0 mg. per lb. body weight) and promethazine (0.5–1.0 mg. per lb. body weight). The total fluid per day was calculated at 60–80 c.c. per lb. body weight and was given as a solution containing 0.2% sodium chloride and 5% invert sugar. To this was added chlorpromazine (2–6 mg. per lb. body weight), promethazine (1.0–2.5 mg. per lb. body weight) and pethidine (1.5–5.0 mg. per lb. body weight). Paraldehyde was given in addition in doses of 1 c.c. by intramuscular injection every 8–12 hours, if the intravenous therapy did not control the spasms. Penicillin was given intravenously in doses of 1,000,000 units per day. If the rectal temperature fell below 94° F., the patient was warmed with an electric pad.

The drip was discontinued after 5–8 days and replaced by naso-gastric feeding, usually of expressed breast milk. The dosage of sedatives and relaxants was reduced at this stage.

FINDINGS

There were 27 males and 25 females (sex not recorded in one) in this series. Of the 53 cases, 31 were admitted during the summer months (October to March) and 22 during the winter months (April to September).

The relationship of the incubation period to mortality rate is analysed in Table 1. Of

TABLE 1: THE RELATION OF THE INCUBATION PERIOD TO MORTALITY RATE.

	Incubation Period		
	6 Days or less	*7 Days*	*8 Days or More*
Number of Patients	33	10	8
Deaths	30 (90·9%)	6 (60%)	3 (37·5%)

Note: The incubation period was not known in 2 cases (1 death; 1 recovery).

the 33 infants with an incubation period of 6 days or less, only 3 survived (mortality rate, 90.9%); 4 of 10 babies with an incubation period of 7 days survived (mortality rate, 60%); while 5 of 8 babies with an incubation period of 8 days or more survived (mortality rate, 37.5%).

Table 2 shows an analysis of the incubation periods in Groups A and B. Among the fatal cases, the average incubation period was 5.5 days in Group A and 6.2 days in Group B. Among the survivors the average incubation period was 8.1 days and 6.6 days for Groups A and B respectively.

The weights on admission ranged from 3 lb. 2 oz. to 13 lb., with an average of 6 lb. 8 oz. Six infants weighed 5½ lb. each or less and they were therefore classed as premature infants. One of these infants in Group B who weighed 5 lb. 4 oz., survived.

Clinical sepsis of the umbilical cord was noted in half the infants. *Clostridium tetani* was isolated in 2 patients.

Of the 53 cases 40 died, the overall mortality rate being 75.5%. This includes patients who died shortly after admission to hospital. Also included in the series is an undoubted case of tetanus (in Group A) with very mild symptoms and signs. This child was admitted 14 days after the onset of symptoms and it was possible to discharge him after 2 days of treatment.

There were 8 survivors among the 28 cases in Group A (mortality rate, 71.5%) and 5 of the 25 infants in Group B survived (mortality rate, 80%). It should be noted that in Group A, only 2 of the 18 fatal cases (11%) lived for a period of 5 days or more, whereas in Group B, 6 of 18 infants (33%) survived for 5 days or longer, 3 of these babies surviving for periods of 8 to 14 days, when spasms had almost ceased.

Evidence of overhydration was noted a day or two before death in 3 of the 6 infants in Group B who lived for 5 days or longer. Electrolyte disturbances were responsible for the death of the fourth infant. Electrolyte levels had been normal on the fourth day of treatment, but when the child collapsed on the sixth day and vomited 'coffee-ground' material, it was found that the serum potassium level had risen to 7.7 m.Eq., per litre. The child had received 0.5 g. potassium chloride in the drip every day (i.e. 6.8 m.Eq. of K per 24 hours), since admission to hospital. Of the remaining 2 patients, one became hypothermic shortly before death (rectal temperature 89.2° F.). At autopsy an extensive bronchopneumonia was found. The other infant died shortly after a rectal temperature of 108° F. had been

TABLE 2: MORTALITY RATES, INCUBATION PERIOD, DURATION OF SYMPTOMS BEFORE ADMISSION AND PERIOD OF SURVIVAL IN HOSPITAL IN GROUPS A AND B. (TWO DIFFERENT TREATMENT REGIMENS)

	Group A		*Group B*	
	Deaths	*Survivors*	*Deaths*	*Survivors*
Number	20	8	20	5
Percentage	71·5	28·5	80	20
Incubation Period	5·5 days*	8·1 days*	6·2 days †	6·6 days
Range	4 to 9 days	6 to 11 days	3·5 to 16 days	5 to 8 days
Average duration of symptoms before admission	1·1 days	4·6 days	1·4 days	2·8 days
Period of survival in hospital ..	*Total* 18 ‡		*Total* 18 ‡	
4 days or less	16 (89%)		12 (66·7%)	
5 days or more	2 (11%)		6 (33·3%)	

Group A: Naso-gastric feeds, phenobarbitone with or without paraldehyde.
Group B: Intravenous therapy with chlorpromazine, promethazine and pethidine.
Note: * Incubation period not known in one case.
† If the case with an incubation period of 16 days is omitted, the average incubation period is lowered to 5·7 days.
‡ Two cases have been omitted from each group as the patients died on the day of admission, in 2 instances before therapy could be started.

recorded. At autopsy the lungs showed areas of atelectasis and moderate congestion, while the liver showed mild fatty change and congestion. It should be noted that in 4 of these 6 infants spasms were completely controlled at the time of death.

DISCUSSION

At first sight the results of treatment as judged by the mortality rates are slightly inferior in Group B, treated with the intravenous 'cocktail.' Survivors in Group B, however, were more severely affected than their counterparts in Group A, as indicated by the shorter average incubation period and shorter average duration of symptoms before admission to hospital. Furthermore, it should be noted that a greater number of infants in Group B lived for 5 days or longer, indicating that the 'cocktail' was superior to phenobarbitone in controlling the spasms. The 6 infants in Group B who remained alive for 5 days or more did not eventually die because of uncontrolled spasms, but of factors such as overhydration, hyper- or hypothermia and hyperkalaemia. This experience suggests that with more careful attention to electrolyte and fluid requirements and to temperature regulation the mortality rate could probably be reduced significantly.

Our decision to substitute, in Group B, intravenous fluid therapy for naso-gastric feeding was based on the finding that infants with tetanus often vomited after naso-gastric feeds and that this often caused death. Tompkins[5] came to a similar conclusion. The intravenous route of administration of drugs has the added advantage of reducing the number of external stimuli which precipitate spasms.

We observed that the 'cocktail' was effective in the control of spasms and rigidity for the first 2–3 days, after which some of the infants developed a tolerance towards the mixture. It then became necessary to add paraldehyde as a sedative, but this was rarely required more frequently than once every 8–12 hours. Thus there appeared to be a synergistic effect between the 'cocktail' and the paraldehyde, which contrasted with its use in the group treated with phenobarbitone, where it had to be given as often as 2- to 6-hourly. More recently we have used pethidine with levallorphan tartrate (pethilorfan) to avoid any possible respiratory depressant effect from pethidine alone. There is no doubt that the treatment regimen adopted for patients in Group B is labour saving, effective and relatively simple to carry out.

This series has shown once more that, apart from drug treatment itself, the prognosis of tetanus neonatorum is affected by factors such as the standard of nursing, the incubation period, the onset interval and the weight on admission to hospital. In our series, as in previous ones,[3, 5] there was a direct association between the length of the incubation period and the survival rate. The survival rate was highest if the incubation period was 8 days or longer. The onset interval, i.e. the period which elapses between the appearance of the first symptoms (e.g. failure to suck) and the onset of the tetanic spasm, is accepted to-day as a more reliable prognostic guide than the incubation period.[5, 6, 12] Unfortunately there were insufficient data in our series to analyse this aspect. Instead we used the duration of symptoms before admission to hospital as an index of the severity of the cases. If this was short the prognosis was poor, while if it was longer the chances of survival were greater.

The prognosis of premature infants with tetanus neonatorum is poor and, indeed, 5 of 6 such infants in this series succumbed. The premature infant who recovered was treated with the intravenous 'cocktail.'

Many reports regarding tetanus neonatorum give the impression that improved results are due to new drugs or techniques, whereas these results could often be attributed to better medical and nursing supervision. This does not apply to our 2 treatment groups, where the nursing care was of the same order throughout.

Our results compare favourably with the simple as well as the other more complicated methods of treatment, such as intermittent positive-pressure respiration with muscle relaxants, which has recently been reported from other parts of South Africa.[6, 7, 13] We therefore recommend the regimen of treatment carried out in our Group B infants as the most convenient and effective one to adopt in any busy Paediatric Unit which has to deal with cases of tetanus neonatorum.

SUMMARY

1. Fifty-three cases of tetanus neonatorum admitted to Baragwanath Hospital between May 1951 and July 1959 are reviewed.

2. The overall mortality rate, including those dying within the first 24 hours, was 75.5%.

In Group A (28 infants) where feeding was via a naso-gastric tube and sedation included phenobarbitone, paraldehyde and bromethol, the mortality rate was 71.5%.

In Group B (25 infants), where intravenous fluid therapy was employed in addition to a 'cocktail' containing chlorpromazine, promethazine and pethidine, the mortality rate was 80%.

Thirty-three per cent of the deaths in Group B and 11% of the deaths in Group A occurred after the fifth hospital day, indicating the superiority of the intravenous regimen.

3. The incubation period was once again shown to be a fairly reliable prognostic index. An incubation period of 6 days or less indicated a poor prognosis. The duration of symptoms before admission to hospital also proved a reliable prognostic guide.

4. The intravenous administration of chlorpromazine, promethazine and pethidine as a mixture, with or without additional paraldehyde, is recommended as a simple, effective and labour saving form of treatment.

Intravenous fluids should not exceed 60 c.c. per lb. body weight per 24 hours and serum electrolyte estimations should be carried out at regular intervals.

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CIRRHOSIS OF THE LIVER IN CHILDHOOD

ITS RELATIONSHIP TO PROTEIN MALNUTRITION (KWASHIORKOR)

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Cirrhosis of the liver is rare in childhood except in some well-defined areas where protein malnutrition is common. Brock has shown that in adults the world distribution of cirrhosis coincides with that of protein malnutrition.[1] In animals, deprivation of choline and methionine produces a sequence of fatty infiltration of the liver and cirrhosis.[2, 3]

In this country, protein malnutrition is common in Bantu children. At this hospital 600–800 severe cases are seen annually. The severest cases occur in children under 3 years old. The cause is usually a diet consisting mainly or exclusively of maize. Despite the high incidence of severe protein malnutrition and poor diet, cirrhosis is rare in the Johannesburg Bantu child. Therefore the concept that cirrhosis is caused by malnutrition requires review.

CIRRHOSIS IN CHILDREN WITH A SATISFACTORY NUTRITIONAL BACKGROUND

In economically advanced communities the rarity of cirrhosis in children and adolescents is reflected by the figures of Mallory[4] who, in 2,066 consecutive autopsies on cases in the first 2 decades of life, found 11 instances of cirrhosis; and Rolleston and McNee[5] who

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found 12 examples in 16,000 autopsies on cases under 21 years of age.

The largest series of children with cirrhosis reported is that of Craig, Gellis and Hsia[6] who, in an excellent monograph, describe 98 cases seen over 30 years (1924–1953) at the Children's Medical Centre, Boston, all proved on biopsy or autopsy. They classify 3 major categories:

(*a*) *Obstructive Cirrhosis.* This was the commonest type. It occurred in 60 of 98 patients, 49 of these being cases of congenital atresia of the extra-hepatic bile ducts and only 1 of which was successfully operated on; 39 died of liver failure during the first year of life, most of the remainder dying of bleeding oesophageal varices after 21 months.

They also describe 5 cases due to other causes of extra-hepatic biliary obstruction of which 2 were choledochal cysts, 2 tumours and 1 traumatic interruption of the common bile duct.

They describe 7 cases of intrahepatic obstruction, all associated with fibrocystic disease of the pancreas (mucosis or muco-viscidosis). None of their cases was due to congenital absence of the intra-hepatic bile ducts, an entity described by other authors.[7] Craig *et al.*[6] and other authors[8, 9] state that the cirrhosis of fibrocystic disease is due to filling of the small bile ducts with thick inspissated eosinophilic material, resembling that found in the pancreatic ducts in this disease. An obstructive dilatation of the bile ducts results, and ultimately causes biliary cirrhosis. The development of cirrhosis in these cases is slow and insidious and never occurs before 6 years of age. In most cases cirrhosis is an incidental finding at autopsy, when death has occurred following the malabsorption syndrome or chronic respiratory infection. In only one of the cases described was cirrhosis the cause of death.

(*b*) *Cirrhosis Associated with Hepatitis.* Craig *et al.*[6] describe 30 cases following neonatal hepatitis. In a separate prospective study the same authors found that 27% of all cases of neonatal hepatitis develop cirrhosis, a much higher incidence than that following infective hepatitis in children. They attribute this high incidence to the following:

i. The infection occurs in the neonatal period when the liver is immature and tolerates infection badly; and

ii. Because it is often difficult to distinguish neonatal hepatitis from congenital atresia of the bile ducts, many are mistakenly operated on with the additional strain of surgery and anaesthesia contributing to hepatic damage.

Human volunteer experiments strongly suggest that the aetiological agent of neonatal hepatitis is the virus of homologous serum jaundice.[10]

Peace,[7] in an analysis of 32 cases of fatal hepatitis and cirrhosis in infancy, includes what is termed 'cholangiolitic' cirrhosis in the group of cirrhosis following viral hepatitis, a condition originally described by Rössle.[11] In these cases, although the extra-hepatic biliary tree is patent, the outstanding feature at autopsy is the presence of large numbers of newly-formed bile ducts. The portal areas are widened and usually linked by fine fibrous septa to efferent veins within the lobules. Under the heading of 'subacute viral hepatitis of unknown aetiology' Peace[7] separates a group of cases in which the outstanding feature is the presence of intra-nuclear inclusion bodies in the liver cells, associated with an extreme giant liver cell reaction. The inclusions resemble those seen in herpes simplex. In addition, in his series of cases, there are children dying of post-hepatitic cirrhosis, following transfusions of whole blood or plasma. These present the same pathological appearances as seen in adults. Congenital syphilis is now a rare cause of cirrhosis; the characteristic histological finding is patchy interstitial fibrosis which may be associated with the formation of multinucleated giant liver cells.

(*c*) *Miscellaneous Causes.* Craig *et al.*[6] describe 7 cases; 2 were due to glycogen storage disease of the liver presenting in the neonatal period; 2 were associated with galactosaemia presenting in the first year of life; 1 followed prolonged cardiac failure; 1 was due to cholangitis and 1 due to haemosiderosis following repeated blood transfusions over a period of 8 years for aplastic anaemia. Hsia *et al.*[12] did not see any cases of cirrhosis following Rh incompatibility and conclude that this condition is not a cause of cirrhosis. They are supported in this by Gerrard[13] who for several years followed 79 children who had had varying degrees of Rh haemolysis in the neonatal period and none of whom had progressed to cirrhosis. Henderson,[14] however, describes cirrhosis following the more severe forms of erythroblastosis and, in particular, demonstated cirrhosis in stillborn infants who had had severe hydrops foetalis.

Peace[7] includes portal or umbilical sepsis as a cause of bacterial hepatitis which may be followed by post-collapse cirrhosis. In such cases, he describes portal panphlebitis, diffuse subacute and chronic inflammation with submassive lobular necrosis of midzonal origin.

CIRRHOSIS IN UNDER-DEVELOPED COUNTRIES

The following varieties of liver disease in childhood are described as being specific to malnourished communities.

FATTY LIVER

Although fatty liver, hepatic necrosis and cirrhosis, and hepatic neoplasms may be readily produced in the experimental animal by dietary deficiency,[2, 3] it has been difficult to apply these findings to Man.

Some investigators[15, 16] believe that malnutrition is responsible for the development of cirrhosis directly from a fatty liver, whereas others[16] feel that under-nutrition simply predisposes the liver to injury and that the fatty liver of kwashiokor although not developing directly into a cirrhotic liver, may remain functionally inadequate and rendered more susceptible to other injurious factors later in life. The latter may be responsible directly for the development of cirrhosis.

In certain diseases of Man which produce a state of secondary malnutrition, a sequence of fatty infiltration and cirrhosis may be demonstrated. Uzman and Hood[17] have shown that in the De Toni-Fanconi syndrome with chronic amino acid loss in the urine, fatty infiltration of the liver occurs and then progresses to cirrhosis. It is suggested[17] that in Wilson's disease, the primary defect is not cirrhosis, but also a chronic amino-aciduria and the cirrhosis is secondary to this. Webster and Williams,[18] discussing the cirrhosis of fibrocystic disease, feel emphatically that this is not an intrahepatic obstruction, but cirrhosis secondary to the chronic malnutrition of malabsorption. They quote the case of a child suffering from fibrocystic disease showing fatty infiltration preceding cirrhosis.

Trowell, Davies and Dean[19] state that an appearance resembling mild Laennec's cirrhosis may occur in older children who have been denied the benefit of treatment for kwashiorkor, and who have passed through the fatty liver stage for a prolonged period.

Waterlow[20] also shows cirrhosis as the climax of prolonged fatty infiltration in children in the British West Indies.

Leevy[16] claims that the lack of documented evidence from liver biopsies in kwashiorkor that hepatic steatosis is a precursor of the fibrotic liver, would make it necessary to seek explanations other than malnutrition for this disease. He is of the opinion that, in adults at any rate, continued poor dietary habits which initially produced fatty liver will ultimately lead to fibrosis.

Higginson *et al.*[21] point out that the age gap between the fatty liver of kwashiorkor and the appearance of the more severe degrees of diffuse portal fibrosis, indicates that the latter is not a direct sequel of kwashiorkor. While regarding the fatty liver of kwashiorkor as a true example of human liver disease due to inadequate diet, they found no evidence that progressive fibrosis, cirrhosis or other sequelae develop directly in such livers. These findings are similar to those of Brock,[1] and Gillman and Gillman.[22] The latter state that the fatty liver in malnourished infants and adult Africans, *per se*, plays no direct role in promoting the high incidence of hepatic fibrosis in Johannesburg.

Rhodes[23] finds little evidence of fibrosis following uncomplicated kwashiorkor and Back and Bras[24] fail to find any fibrosis after 3½–5

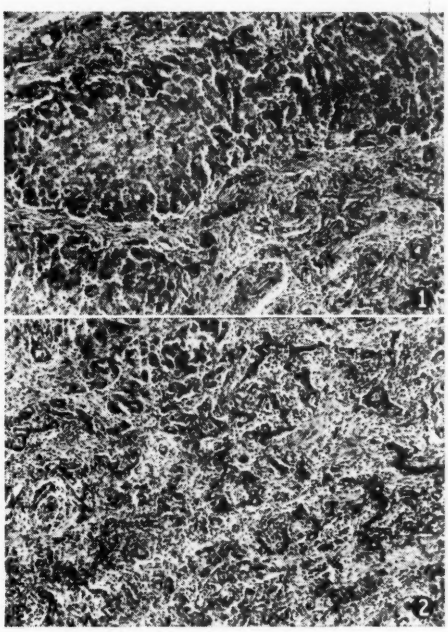


Fig. 1. Section of the liver to show well-marked biliary cirrhosis. Islands of regenerating liver cells are surrounded by thick bands of collagenous tissue containing large numbers of proliferating bile ducts. Bile stasis is a prominent feature.

Fig. 2. Intense proliferation of bile ducts with mild lymphocytic infiltration.

years follow up in 10 cases of kwashiorkor which had previously shown fatty livers on biopsy.

CIRRHOSIS IN JAMAICA

Veno-occlusive disease of the liver is seen in Jamaica and may progress in the late stages to centrilobular cirrhosis.[25-27] This condition occurs most commonly between the ages of $1\frac{1}{2}$ and 3 years and it is in this period or later that cirrhosis may supervene. The children affected are usually poorly nourished, although rarely cases of kwashiorkor. Fatty infiltration is seen in Jamaica, as elsewhere, in association with malnutrition, but is seldom seen in cases of veno-occlusive disease.

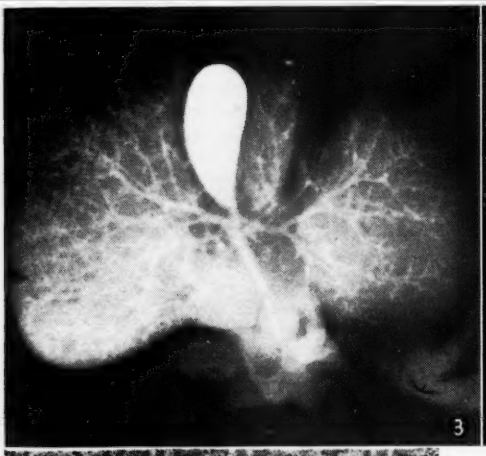
Selzer and Parker[28] report a series of cases of senecio poisoning where a picture resemblig veno-occlusive disease is demonstrated. The senecio in these cases was ingested by eating bread made of inadequately winnowed wheat. More recently Hill *et al.*[29] clearly demonstrated the production of veno-occlusive disease in experimental animals by giving them crotallaria.

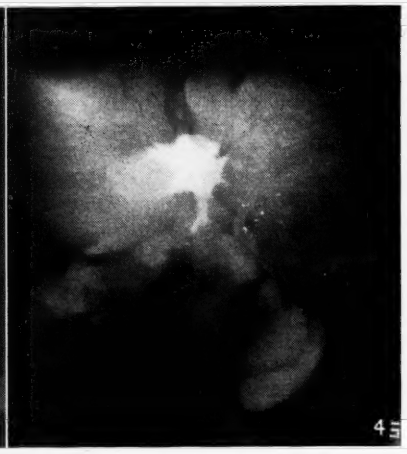
INDIAN CHILDHOOD CIRRHOSIS

Indian childhood cirrhosis is also largely confined to a specific geographical area and has stimulated much investigation and writing about its relation to malnutrition.

The Indian Council of Medical Research[30] states that 'infantile cirrhosis is a disease peculiar to India, affecting infants and young children, with a tendency to run in families.'

Chanda[31] in a recent paper claims to have shown the evolution of cirrhosis from fatty change by studying the histology of the liver in children suffering from kwashiorkor. He describes 4 such fully developed cases of cirrhosis in children under 4 years of age one





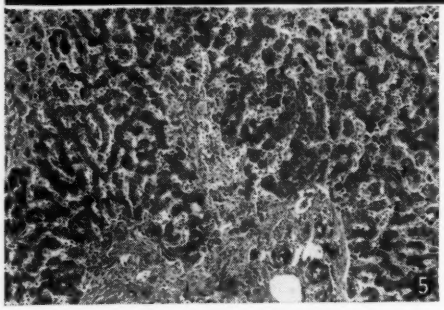


Fig. 3. Post-mortem injection study of normal biliary tree.

Fig. 4. Post-mortem injection study of biliary tree in a case of congenital agenesis of the intrahepatic bile ducts. Note complete absence of dye in the liver with filling of the rudimentary gall bladder only.

Fig. 5. Photomicrograph of the liver shown in Fig. 3. Bile ducts are absent from the portal tracts and bile stasis is prominent.

The Jamaican workers have long suspected a toxic etiology, particularly in view of the vast quantities of bush teas that are imbibed by the Jamaican indigents. These contain a variety of alkaloids, among them senecio and crotallaria. It is well-known that senecio poisoning in cattle causes a cirrhotic condition which resembles veno-occlusive disease.

of whom was 5 months old. He states, however, that there may be other factors in addition to fatty infiltration which may help the progression to cirrhosis but 'what these are we do not know.'

Singh *et al.*[32] describe children of middle and lower-middle class Indian families who had cirrhosis. They state that none of their cases had a histological picture resembling veno-occlusive disease and suggest that the aetiology is either hereditary or due to virus infection.

Jelliffe *et al.*,[33] investigating Indian childhood cirrhosis, point out the difficulties of establishing a clear pathology in these cases because of difficulties in obtaining autopsies. They quote some of the Indian writings suggesting an association of Indian childhood cirrhosis and veno-occlusive disease. Six cases which they investigated by needle biopsy or at autopsy showed some involvement of central veins and therefore tended to confirm this view. They suggest that where investigations have shown patent central veins, these may be due to collateral circulation which can occur in veno-occlusive disease.

THE SOUTH AFRICAN BANTU CHILD

In the Paediatric Department of Baragwanath Hospital, where we admit about 5,000 Bantu children under the age of 10 years to our wards annually, and where about 200 paediatric autopsies are done annually, cirrhosis of the liver is rarely seen. The potential and established cases of cirrhosis which we do see, conform largely to the pattern seen in children with a satisfactory nutritional background.

Two to three cases of congenital extrahepatic obliteration of the bile ducts are admitted annually. Most of these have exploratory operations and are then discharged. Due to difficulties of follow-up only 2 of these patients have been watched to the terminal phase of cirrhosis; autopsy was performed on one of them, an 8 month old child shown to have biliary cirrhosis (Figs. 1, 2). One case of congenital intrahepatic obliteration of the bile ducts has been encountered (Figs. 3–5). We have also seen an isolated case of xanthomatous biliary cirrhosis presenting with obstructive jaundice, xanthomatous nodules on the body, and a very much raised serum cholesterol value. Fibrocystic disease of the pancreas has never been recognized in a Bantu child here, although we are very conscious of this condition. We have therefore never seen biliary cirrhosis due to this disease.

Neonatal hepatitis is not uncommon in children at this hospital, about 6–8 cases being diagnosed annually. Again, due to difficulties of follow-up, the incidence of complicating cirrhosis is uncertain. Recently a 3-day-old child with intense jaundice died and was found at autopsy to have cirrhosis due to neonatal hepatitis (Figs. 6, 7). We have seen one autopsy-proved case of cholangiolitic cirrhosis in a 3½-year-old child admitted in hepatic coma and there have been 3 cases of 'cholangitis.'

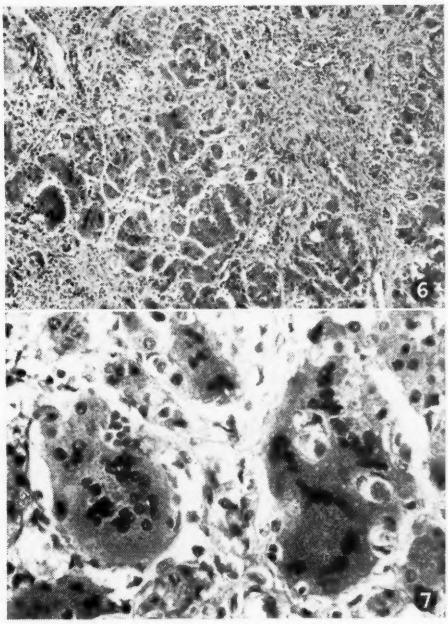


Fig. 6. Section of the liver from a case of cirrhosis due to neonatal hepatitis. Thick collagenous septa break up the liver lobule and there are numerous giant liver cells.

Fig. 7. Higher magnification (four-fold) of Fig. 6 to show the giant liver cells. The granules in the cytoplasm are haemosiderin.

We have never found any instances of cirrhosis due to glycogen storage disease of the liver or galactosaemia. Many cases of cardiac failure due to congenital and rheumatic heart disease, cor-pulmonale, pericarditis and idiopathic cardiac hypertrophy are seen, but established cardiac cirrhosis has never been found.

Bilharzia is reported to cause cirrhosis of the liver, particularly in Egypt.[34, 35] This parasitic disease is endemic in and around Johannesburg, but Higginson and de Meillon[36] (in-

vestigating it locally) conclude that in this area there is no significant correlation between liver fibrosis, cirrhosis and the presence of schistosomiasis. They are supported in this view by other investigators.[37-39] In 1954 we admitted a 5-year-old child who had cirrhosis, which was considered to be of bilharzial origin on the basis of a bilharzial ovum found on liver biopsy. However, this child died after readmission last year, and was found to have post-necrotic cirrhosis.

Positive serology for syphilis is not uncommonly found in the mothers of children born at this hospital, but clinical manifestations of congenital syphilis in these children are uncommon. Neonatal jaundice attributable to syphilis is a rare phenomenon here, and our autopsy series contains only one case of diffuse interstitial fibrosis of the liver due to congenital syphilis.

In the past 4 years sporadic cases of veno-occlusive disease of the liver have been encountered in children here,[40] 6 having been proved by biopsy or autopsy. However, all of these have been pre-cirrhotic.

Protein malnutrition occurs abundantly in our community and we admit 600–800 severe cases annually of whom about 20% die. Several hundred have now come to autopsy and though fatty infiltration of the liver is an invariable finding in these cases, we have never encountered a case where this was associated with evidence of cirrhosis. Mild periportal fibrosis with increased reticulin is found in the livers of malnourished children, but there is never architectural distortion of the liver lobule or regenerative hyperplasia.

COMMENT

This survey has shown that in a child population where protein malnutrition is common, the incidence and causation of cirrhosis of the liver do not differ fundamentally from those encountered in overseas populations, with a vastly superior nutritional background.

There is no doubt that, in some populations, cirrhosis of the liver occurs in association with severe protein malnutrition, even in childhood. In view of our findings it seems doubtful whether the two conditions are causally related. However, it should be remembered that in this part of Africa protein malnutrition is usually caused by a diet consisting mainly of maize, and that other deficient diets with different amino acid patterns, may cause liver damage of a different nature. Our survey, therefore, does not exclude the possibility of the existence in children of cirrhosis, which is truly dietetic in origin. Nevertheless, at least in one country where protein malnutrition is prevalent, childhood cirrhosis of the liver has been traced to a toxin consumed in the generally inadequate diet. It is not unlikely that the curious geographic distribution of childhood cirrhosis will eventually be found to be due to hitherto unrecognized toxins.

SUMMARY

The incidence of cirrhosis in children with a satisfactory nutritional background is compared with that of children in under-developed countries.

Apart from some specific geographical areas, the incidence is similar despite varying nutritional backgrounds.

It is suggested that where childhood cirrhosis is common in under-developed countries, the aetiological factor may be some toxin in the diet, as has been proven in Jamaica, and that malnutrition *per se* does not cause cirrhosis in childhood.

We would like to express our thanks to the Superintendent for permission to submit this paper for publication; to Drs. E. Kahn and S. Wayburne for advice and access to case records; and to the Director of the South African Institute for Medical Research for facilities granted.

Mr. M. Ulrich of the photographic department of the South African Institute for Medical Research produced the photographs.

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THE BARAGWANATH HOSPITAL DIABETIC CLINIC

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and

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Baragwanath Hospital, Johannesburg

> 'Diabetes is a wonderful affection . . . being a melting down of the flesh and limbs into urine . . . thirst unquenchable; excessive drinking, which however is disproportionate to the large quantity of urine.'
>
> —*Aretaeus,* 2nd Century.[1]

Diabetes mellitus is reputedly rare in the African. In Johannesburg, however, it is a common and important clinical problem. This has been recognized only in recent years, and knowledge concerning its management is as yet meagre. A Diabetic Clinic was therefore established at this hospital in July 1958. It is hoped that this report will encourage the establishment of similar clinics amongst other African population groups and provide advice in practical management.

Since its inception 115 patients have been referred to the clinic and these represent but a fraction of the total number of diabetics receiving treatment at the hospital. The patients are an unselected group, being referred at random from the wards, where their diagnosis and initial stabilization are undertaken.

Three quarters of our patients are over the age of 40 years. Of the remainder, most are between the ages of 21 and 40. Only 3 patients are under the age of 20, the youngest being 13 years old. The sex distribution shows a 3 : 2 female predominance. Two thirds of the patients are obese or overweight, most of these being middle-aged or elderly. The remainder are either of average build or thin and are more or less evenly distributed between the different age groups. The 3 patients under the age of 20 are all thin.

At the first attendance each patient is questioned and examined according to a fixed

scheme (Appendix 1). The frequency of subsequent visits is determined by the severity of the illness and the ease of control. An attempt is made to see each patient at least one a month.

The clinic is held on a Saturday morning. This has been found to be the most convenient time for most African patients and has yielded a follow-up rate of 75%. At each visit the patient is weighed, his urine examined for sugar, acetone and albumin and specific inquiry is made into frequency of micturition, the presence or absence of thirst and the development of any other untoward symptoms. Every patient is fitted with an irremovable wrist-bracelet marked 'Diabetic.'

MANAGEMENT

We believe that most of the serious effects of diabetes are due to water loss from the body. This may amount to many litres in 24 hours. In consequence inquiry about frequency of micturition is more important than estimating the amount of sugar in the urine.

If frequency of micturition is within normal limits on the one hand, and there is no depression of either neurological function or mental ability to indicate hypoglycaemia on the other, we are satisfied that the patient is controlled.

We would emphasize that attempts at rendering the urine sugar-free in the illiterate insulin-treated African diabetic are not only impracticable but also dangerous, since they are liable to result in hypoglycaemia. This, in view of the undue delay which most African patients experience in obtaining medical attention, is not infrequently fatal, or gives rise to permanent brain damage.

Insulin. Three quarters of our patients are on insulin, which is self-administered. During their stay in the wards the patients are taught the use of a syringe. On the whole they have adapted themselves well to the injection technique and septic or other complications have been remarkably uncommon. We have had only one injection abscess during the past year. All our patients are stabilized on one daily injection of a long-acting insulin, usually of the *lente* type. Dosage is checked on the patient's own syringe which he brings to the clinic at each visit.

Diet. Since the diet of the African is predominantly carbohydrate, the more usual medical attitude to dietary control of the African diabetic has been frankly pessimistic. Consequently, it has been the custom in the past to treat all diabetics with insulin in amounts designed to cover the high carbohydrate intake —a clearly undesirable situation considering that two thirds of the cases are overweight.

At the clinic we have made a renewed effort to achieve dietary control and have thus far met with a surprising degree of success. In large measure this has been due to the efforts of the Senior Dietician, who has undertaken a detailed analysis of the patients' diets. Twenty-six patients who had been receiving an average of about 40 units of insulin daily have been rendered symptom-free by diet alone. In most of these the urine has also become sugar-free. This has been achieved not so much by an absolute reduction in carbohydrate intake, as by redistributing the carbohydrate intake throughout the day. The urbanized African usually consumes the bulk of his high carbohydrate diet with the evening meal. In the diabetic this must result in nocturnal polyuria. In fact, all our patients when first seen complained of nocturnal frequency, which was little influenced by their single morning dose of insulin. By redistributing the carbohydrate intake so that the major portion is consumed during the day, when physical activity is greatest, nocturnal frequency is rapidly reduced. In these circumstances we have found that household measures (the plateful of 'this', the slice and the spoonful of 'that') provide a satisfactory balance.

The benefits of this simple device are so immediately obvious to the patient that there is then little difficulty in obtaining his continued co-operation in maintaining the dietary regime. A few patients who were taking 3 large carbohydrate meals a day have been persuaded to reduce the evening meal drastically, with similarly beneficial results.

In view of our success with dietetic therapy we have not used oral anti-diabetic drugs.

Finally, as some index of the overall efficacy of control, we would mention that since the inception of the clinic, only 4 patients have required readmission to hospital—2 with infections (paronychia and vulval abscess), 1 aged 15 years with recurrent episodes of diabetic pre-coma, and 1 for re-stabilization. There were no deaths.

We are grateful to Dr. V. H. Wilson, Senior Physician, Baragwanath Hospital, for encouragement and advice in the preparation of this paper.

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APPENDIX 1

DIABETIC CLINIC

Name

Address

Age Sex

Marital Status Race

Occupation Wage

Educational Level

Birthplace and Duration of Stay in Jhb.

Attendance at Witch-doctor

Diet and Cooking Utensils

Alcohol: Type and Quantity

Method of Presentation and How Referred to Clinic

Stabilization and Duration of Stay in Hospital

Physical Examination
Blood Pressure *Height* *Weight*

Previous Illnesses and Hospital Admissions

(Especially ketosis and hypoglycaemic coma)

Family History

COMPLICATIONS AND INVESTIGATIONS

Eyes
Eye-lids
Cornea
Pupils
Lens
Retina
Glaucoma
Refractive Error

Skin
Pruritus
Xanthomata
Carotenaemia
Dupuytren's
Pyogenic
Fungal
Allergy
Insulin Lipodystrophy

Vascular System
Angina
Claudication
Trophic
Colour
Pulses
Temperature
Oedema

Respiratory System
Pyogenic
Tuberculous

Nervous System
Cranial Nerves
Motor System
Sensory System
Reflexes
Vibration Sense

Digestive System
Teeth
Jaundice
Colic
Dyspepsia
Nocturnal Diarrhoea

Renal Tract
Albuminuria
Pyuria
Retention
Precipitancy

Gonads
Male: Impotence
Fertility
Female: Menses M D L
Parity
Gravidity

Endocrine Glands
Acromegaly
Cushing's
Thyrotoxicosis

Diabetes and Pregnancy
Number of Pregnancy 1 2 3 ... 11 12
Oedema
Albuminuria
Hypertension
Abortion
Prematurity
Dystocia
Infant—Size
—Congenital Abnormality
—Fate
Lactation
Effect of Pregnancy on Diabetes

Special Investigations
(1) Blood Count and Sedimentation Rate
(2) Serum Proteins
(3) Urea
(4) Lipid Studies
(5) Glucose Metabolism
(6) X-Ray of Chest and Abdomen
(7) Microscopy and Culture of Urine

FOOD SERVICE TO PATIENTS AT BARAGWANATH HOSPITAL IN RELATION TO NUTRITIONAL ADEQUACY, ACCEPTABILITY AND COST

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Principal Dietitian, Baragwanath Hospital, Johannesburg

Baragwanath Hosital caters for over 1,600 patients—medical, surgical, paediatric, gynaecological and obstetrical cases. In addition there are extra stretchers placed in most wards, and many out-patients are kept unavoidably for long periods at the hospital. In the paediatric out-patients' department alone 3,029 infants and 3,029 mothers slept over in this unit for the first six months of the year. These out-patients are not reflected in the bed state of the hospital so that the numbers being catered for are almost 2,000.

The patients are of varied nationalities. There are Basutos, Bapedis, Nyasas, Shangaans, Swazis, Tswanas, Vendas, Zulus, Xosa, Indians —some of whom are Mahommedans, others Hindus. There are also coloured patients.

Most of the inmates come from the locations where they have resided for a number of years. Few have retained their former food habits. Many have never known tribal life nor have they lived in a rural environment. Most of them have adopted, or are in the process of adopting, the European food pattern.

The diets served in Baragwanath Hospital are full ward diet (1,000), light diets (300), therapeutic diets (250), liquid diets and supplementary liquid feeds (200). There are over 200 infant patients (exclusive of infant out-patients), for whom milk feeds are prepared.

The diets (appendix) conform as far as possible to the food customs of most non-Europeans. They are simple because the organization in the dietary department is such that there is no time for complicated procedures. Diets are standardized as far as possible to eliminate unnecessary equipment and labour. In like manner, the particular foods served are reduced to those which are generally acceptable to the greater number of patients.

Care is exercised in the department in the requistioning of provisions in relation to requirements and cost, in preparation as to wastage by inferior cooking procedures, and in service by supplying only actual requirements.

As in all Hospital Dietary Departments the general food pattern is modified in consistency and nutrients. The latter is necessary in all patient treatment more so in Baragwanath Hospital where so many of the patients have been previously undernourished.

An analysis of the full ward diet is given in terms of calories, protein, calcium, iron, vitamin A, thiamin, riboflavin, niacin and ascorbic acid. By comparison with the Recommended Minimum Daily Dietary Standards as compiled by the National Nutrition Council of South Africa, the full ward diet of Baragwanath Hospital is *not* excessive.

In relation to the recommended dietary standards attention has been given to the following:

1. The standards give minimum requirements for whites and non-whites and are drawn up to suit the mixed population of South Africa with its varying food habits.
2. The allowances do not make provision for additional requirements during illness nor defects in absorption and metabolism.
3. The Council advises that the standards be used with discretion. They are however valuable as a guide in assessing the nutritional adequacies of a feeding regime.

The analysis of the Baragwanath full ward diet must be viewed in the light of the following:

1. An analysis computed from food value tables cannot be regarded as completely accurate.
2. Although allowances were made for losses of minerals and vitamins in cooking, it is possible that food as served would have lower values than these presented. This relates particularly to ascorbic acid values.
3. Protein values have to be regarded in terms of amino acid content and not as total protein. Fifty per cent. of the protein of these diets is provided from foods of plant origin. Patients protein requirement should be high in proteins of high biological value that is as obtained from animal and dairy products.
4. Vitamin A values include carotene a precursor of Vitamin A. It is recommended that at least one third of the Vitamin A requirement should be present as such—that is as obtained from dairy products and animal fat. Failing this the requirement of Vitamin A should be higher than the given standards.
5. Calcium values of the Baragwanath diets appear to be high. It is to be noted however that much of the calcium has been supplied by cereals. Phytic acid present in cereals precipitates calcium from the food as well as affecting absorption of other calcium in the tract.

In the treatment of medical cases the use of bulky, gaseous-forming foods is contra-indicated. The diets of such cases are compiled from readily digestible forms of meat, fresh vegetables, milk and eggs.

The treatment of malnutrition and avitaminosis calls for a high intake of all nutrients. As some factors are known to exist in natural foods but have not been isolated, physicians prefer to treat such patients with diets compiled from a variety of nutritious foods. Hence, the need for the use of meat, fresh fruits and vegetables and dairy products.

The surgical cases' requirements of all nutrients are higher than normal. Because of trauma, additional protein of high biological value is needed to replace body tissue. Increased vitamins are required for the same reason. Paraplegic patients are treated with high protein diets. Severe burn cases have need of increased protein and vitamins. Fracture cases depend on protein to assist in the absorption of calcium required for healing broken bones.

It has been shown that post-operative recovery is influenced by the nutritional state of the patient. *Wounds heal more quickly, convalescence is shortened and there is a quicker turn-over of hospital beds.*

The full ward diet is used mostly for convalescing surgical patients, malnutrition and avitaminosis cases, the latter being supplemented with fortified milk feeds.

The feeding costs for all patients in Baragwanath Hospital are approximately 2s. per patient per day as compared with 2s. 6d. in a similar, but not as large, non-European Hospital in the Transvaal. Food expenditure in a European Hospital has been estimated as 7s. per patient per day.

The physician, surgeon and paediatrician rely on the hospital dietitian to provide an adequate diet for all patients confined to their care. They realize the importance of the various nutrients in the treatment of their medical, surgical or paediatric cases.

Whilst it is the aim of the dietitian to plan diets in relation to foods habits, religious customs and money available for food expenditure, she is not practising her profession if she is ignoring the adequacy of the diets being served. It is possible, however, within reason, to give attention to all four such factors in planning meals for patients and such is the policy of the Dietary Department, Baragwanath Hospital.

APPENDIX

BARAGWANATH HOSPITAL DIETS

	Full Ward Diet (1,000)	*Light Diet* (300)	*Liquid Diets* (±200)
Breakfast	Mealie meal porridge with milk and sugar. Slice enriched bread. Coffee with milk and sugar.	Mealie meal porridge with milk and sugar. Slice white bread.	Hourly or two hourly milk feeds and feeds consisting of milk fortified with milk powder, cream and eggs. Strained soup. Jelly and thin milk puddings. Orange juice. Thin porridge.
Mid a.m.	Slice enriched bread with jam. Tea with milk and sugar.	Slice white bread with smooth jam. Tea with milk and sugar.	
Dinner	Meat as braised or stewed. Dried peas or beans. Mealie rice or samp. Fresh vegetables such as cabbage, pumpkin or carrots. Milk pudding. Fresh fruit.	Strained soup. Minced meat or tripe. Potatoes, pumpkin or carrot (pureed). Milk pudding. Custard. Fresh fruit.	*Therapeutic Diets* (250) The diets in this group include those prescribed for such conditions as cardiovascular diseases, diseases of the gastro-intestinal tract, diabetes, liver and kidney disorders and typhoid fever.
Mid-Afternoon	Slice enriched bread with butter or (margarine). Tea with milk and sugar.	Slice white bread with butter or (margarine). Tea with milk and sugar.	
Supper	Kaffir corn porridge. Thick soup prepared from minced vegetables and mince meat. Slice enriched bread.	Kaffir corn porridge. Mince with potatoes or egg. Slice white bread. 8 *p.m.* Cup hot milk.	*Infants* (200) Almost entirely milk feeds.

RECOMMENDED MINIMUM DAILY DIETARY STANDARDS[1]

Compiled by Dietary Standards Committee National Nutrition Council of South Africa

	Calories (*g.*)	*Protein* (*g.*)	*Calcium* (*g.*)	*Iron* (*mg.*)	*Vitamin A* (*Intravenous*)	*Thiamin* (*mg.*)	*Riboflavin* (*mg.*)	*Niacin* (*mg.*)	*Ascorbic Acid* (*mg.*)
Man (Average weight 160 lb.), moderately active	3,000	65	0·7	9	4,000	1·0	1·6	15	40
			Full Ward Diet Baragwanath Hospital						
Average Adult Patients	3,000	110	1·2	27·8	5,529	1·5	2·26	19	130

1. S. Afr. Med. J., Vol. **30**, 4 February 1956.

RECORDS OF DIAGNOSED CASES OF DEFICIENCY DISEASES OR DISEASES WHERE MALNUTRITION IS CONSIDERED A CONTRIBUTING FACTOR

	1951	1952	1953	1954	1955	1956	1957	1958
Tuberculosis	903	980	1,183	1,011	1,139	1,054	1,246	1,217
Pellagra	174	171	223	279	197	184	272	243
Malnutrition	340	395	488	658	714	747	747	963
Rickets	19	46	52	62	100	111	109	129
Scurvy	1	5	33	34	19	23	28	33
Beri-beri	2	–	7	–	10	12	13	11
Atrophy of the liver	7	16	12	13	12	8	11	13
Cirrhosis of the liver	95	64	64	89	91	100	112	131

PLEURAL BIOPSY IN THE DIAGNOSIS OF PLEURAL EFFUSION

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An aetiological diagnosis is frequently not established in patients with pleural effusion. Biopsy of the parietal pleura under direct vision has been performed for diagnostic purposes.[1–3] This procedure, however, is a surgical one and possesses disadvantages. De Francis *et al.*[4] were the first to report on needle biopsy of the pleura utilizing a Vim-Silverman needle. This method of aspiration biopsy of the pleura was also employed successfully by Heller *et al.*,[5] Donohoe *et al.*[6] and Weiss.[7]

Abrams[8] in 1958 described a pleural biopsy punch which enabled a biopsy specimen to be taken from the parietal pleura, without risk of damage to the lung. Mestitz *et al.*[9] have related their experience with this punch,

mostly in the diagnosis of tuberculous effusions.

We report here our results of biopsy of the parietal pleura using the punch described by Abrams.[8]

CLINICAL MATERIAL AND METHODS

Pleural biopsies were carried out in 50 Bantu patients, most of whom had a pleural effusion at the time of the investigation. In a few patients the procedure was performed in the absence of fluid in the chest in order to elucidate a lung or pleural shadow of unknown origin. Whenever possible, biopsies were done before commencing treatment. The ages of our patients ranged from 21 to 55 years. There were 42 males and 8 females. Other investigations included radiography of the chest, sputum examination for the tubercle bacillus, microscopy and culture of the pleural fluid.

Aspiration of the pleural effusion and biopsy of the parietal pleura is carried out during the same procedure. Fig. 1 shows the features of the punch. It consists essentially of 2 concentric tubes with a stylet. The outer tube has a deep notch near its distal end and the inner tube has a sharp cutting edge. The chest wall is infiltrated with local anaesthetic, a small incision is made into the skin, and the punch, with the notch closed, is introduced into the pleural cavity. The notch is opened and the fluid aspirated. The punch is then slowly withdrawn to the edge of the parietal pleura and a biopsy is taken by advancing the cutting edge of the inner tube. Several specimens of tissue may be obtained by repeating the performance through the same skin puncture.

No complications were encountered and the procedure was no more distressing to the patient than an ordinary chest paracentesis. The potential risks are haemorrhage, pneumothorax, spread of metastases and contamination through introduction of infection. Complications were not encountered by Abrams.[8] Mestitz *et al.*[9] reported the development in one patient of secondary deposits along the needle track 18 months after the biopsy. There is no contraindication to pleural biopsy, although it is not advised in patients with haemorrhagic disease.

RESULTS

Pleural tissue was obtained in 32 patients (64%). Eight of these patients were considered to be suffering from an acute inflammatory disease (pneumonia: 6 cases; pulmonary suppuration: 2 cases). This was confirmed by biopsy, the pleural tissue showing the features of an acute inflammatory lesion. The remaining 24 patients presented with primary pleural effusions without radiological evidence of parenchymal lung disease. Sputum examination for tubercle bacilli was negative in all these cases. Since tuberculosis is a common cause and sequel of primary pleural effusion[10] these patients were regarded and treated as tuberculous pleurisy. Histological examination of the pleural tissue obtained from 12 of these 24 patients revealed the

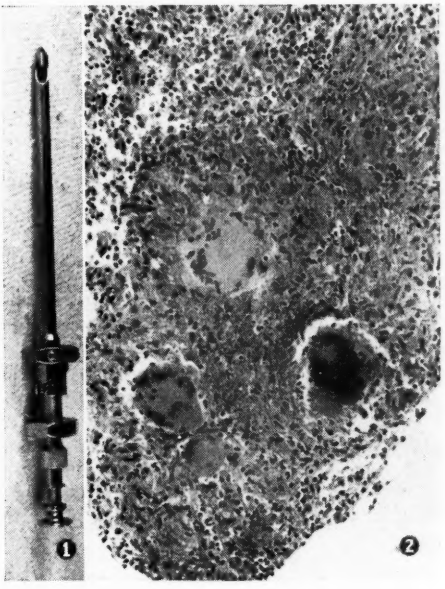

Fig. 1. Pleural biopsy punch of Abrams. Note notch at distal end of outer tube..

Fig. 2. Histological section of the parietal pleura showing tuberculous granulation tissue with epithelioid cells, Langhans-type giant cells and lymphocytic infiltration.

features of tuberculosis (Fig. 2). Acid-fast bacilli were found in 2 of these biopsies, but this is not a true reflection of their incidence as Ziehl-Neelsen stains were not done routinely. Granulation tissue was demonstrated in 4 other biopsies and, although this may indicate tuberculosis, the findings were not considered diagnostic. In the remaining 8 patients histology of the pleura showed the non-specific changes of chronic inflammation and fibrosis.

Biopsy in 18 cases was unsuccessful as the specimen obtained revealed only muscle or connective tissue. Most failures occurred early

in the study and decreased with experience. The ability to obtain pleural tissue depends also on the thickness of the pleura, the presence of free fluid and the number of specimens taken during the procedure. Biopsy is more difficult in the absence of an effusion, as the fluid helps to locate the site of the parietal pleura.

DISCUSSION

There is a definite need for additional diagnostic methods to elucidate the aetiology of pleural effusion. Punch biopsy of the pleura is a simple and safe procedure and may be valuable in obtaining an early diagnosis. Thus, Mestitz *et al.*[9] were able to establish a diagnosis by this method in approximately 80% of tuberculous and 60% of malignant effusions. In our own series we found the procedure helpful in 50% of 24 patients with suspected tuberculous effusions.

It must be stressed that the finding of non-specific changes in the pleura does not exclude the possibility of tuberculosis or malignant disease. In tuberculous pleurisy the pleura is usually diffusely involved, whereas in malignant disease the deposits of malignant tissue may be widely scattered. Biopsy has consequently been found to be less useful in patients with malignant involvement of the pleura than in cases with tuberculosis.[7, 11]

It is recommended that biopsy of the parietal pleura should be done simultaneously with aspiration of the pleural fluid in every patient with effusion of doubtful aetiology, in order to establish an early diagnosis.

SUMMARY

We report our experience with punch biopsy of the parietal pleura in 50 patients.

A tuberculous aetiology was proved in 12 of 24 patients with primary pleural effusion.

The procedure is simple and safe and often gives an accurate and early diagnosis when other methods have failed. It should be part of the initial investigation of every patient with a pleural effusion of uncertain aetiology.

We wish to thank Dr. Isaacson and the Pathology Department for the histological examination of the biopsies and Mr. M. Ulrich, of the South African Institute for Medical Research, for the photomicrograph.

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ARCUATE TYPE OF PIGEON BREAST DEFORMITY

IN BROTHER AND SISTER

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One seldom sees two identically deformed chests in a brother and sister, as in Stephen M., aged 16 (Fig. 1), and Jacobeth M., aged 14 (Fig. 2).

Jacobeth presented herself with a troublesome cough. She was accompanied by her mother and her brother Stephen.

Both these children were healthy, strong, well built, well grown and of good posture. The sternum protruded at the angle of Louis and had, in addition, many features of funnel chest. The thorax was symmetrical, the heart was normal in shape and position (Figs. 3 and 5), and the xiphoid moved freely forward with inspiration. The xiphi-vertebral space was roomy and adequate (Figs. 4 and 6). The abdomen displayed no pot belly and the shoulders were not rounded.

A panoramic view of the chest contour, as seen in the neonate, through childhood to adult, shows the rib cage circular at birth with gradual disappearance of its rotundity from about the third year and becoming ovoid. The xiphi-vertebral diameter diminishes and the

lateral widens until the ellipsoidal shape in the adult is reached. The ribs, which are resilient at birth and early childhood, become firm and rigid in adolescence.

Partly because of the rotundity and resilience of the chest wall in the early years of life congenital and developmental deformities are not often seen. Most of these malformations become manifest with growth and the passage of years. An appreciable number of funnel chests appear at birth or early in infancy. Others appear later with increased growth, as at puberty. Pigeon chests, however, are seldom seen before the age of 6 years.

ETIOLOGY

Heredity plays a significant role in anterior chest wall deformities. The mother of these children is adamant that both the father and paternal grandfather had similar chest deformities. Hence these children illustrate a heredofamilial malformation.

Fig. 1. Stephen, aged 16 years. Arcuate sternum and funnel chest.

Fig. 2. Jacobeth, aged 14 years. Arcuate sternum and funnel chest.

Inherited chest deformity is specific as to *site* but not as to *type*.[1] Thus the ancestor may have an anterior chest wall deformity and likewise the offspring. But the ancestor may have a pigeon breast and the offspring may inherit a pigeon breast or a funnel chest or any other anterior chest wall deformity such as an asymmetrical funnel chest or an oblique protrusion deformity of the sternum.

PATHOGENESIS

In Stephen and Jacobeth there was proportionate *overgrowth* of the second and third pairs of ribs. Buckling of these ribs was inevitable and must occur at their weakest point, the *locus minoris resistentiae* being the costochondral junction. The sternum could either protrude or cave in. The direction depended largely on the diaphragm, which failed to grow *pari passu* with the overgrown ribs. By anchoring the xiphoid process it triggered the sternal protrusion (Figs. 4 and 6). The diaphragm sacrificed part of its dome to accommodate the adventitious arch of the arcuate sternum. The costal cartilages of the corresponding second and third pairs of ribs slanted forward to the elevated sternum at the angle of Louis.

CLASSIFICATION

Based on the pathogenesis, Lester[1] divides congenital and developmental anterior chest wall deformities into 3 groups:

I. Depression deformities (pectus excavatum, funnel chest);

II. Protrusion deformities (pectus carinatum, pigeon breast);

III. Combination deformities.

Groups I and II appear to be exact opposites. Group III comprises combination deformities —a heterogeneous group. It is a concoction of permutations and combinations of diverse funnel chests and pigeon breasts, displaying characteristics of both.

DIAGNOSIS

The described deformity of Stephen and Jacobeth combines clinical features of pigeon breast and funnel chest. This makes the diagnosis difficult. In such a dilemma Lester[1] leans on the *dominant* factor for diagnostic guidance, the dominant factor being the outstanding characteristic in the deformity. Here the most glaring feature is the bulge and arch of the sternum at the angle of Louis.

Lester's carefully considered classification of this anomaly[1] is:

Protrusion deformity (pectus carinatum, pigeon breast);

Midline;

Arcuate sternum.

Albeit unwieldy, it is explicit. The deformity is 'pigeon-holed'. Many of us would have mistaken this condition for a funnel chest (pectus excavatum).

PSYCHOLOGICAL REACTION

The chest is aesthetically important. In the male, a well-formed muscular thorax of good posture is a source of pride. A shapely, well-chiselled chest elegantly cushioned is enchanting in the female. But the unfortunate funnel-chested or pigeon-breasted child is often tormented by sadistic school mates. To avoid exposure of his rib cage deformity, he fights shy of swimming, sunbathing and athletics.[3]

Although the handicap is largely cosmetic, a severe funnel chest may compress and dislocate intrathoracic viscera. The heart may be dislodged into the left hemithorax and the lungs compressed, resulting in dyspnoea on exertion. The major complaint is a non-productive cough. The cough is an essential defensive reflex. For its effective production the thorax must act as a strong pair of bellows.

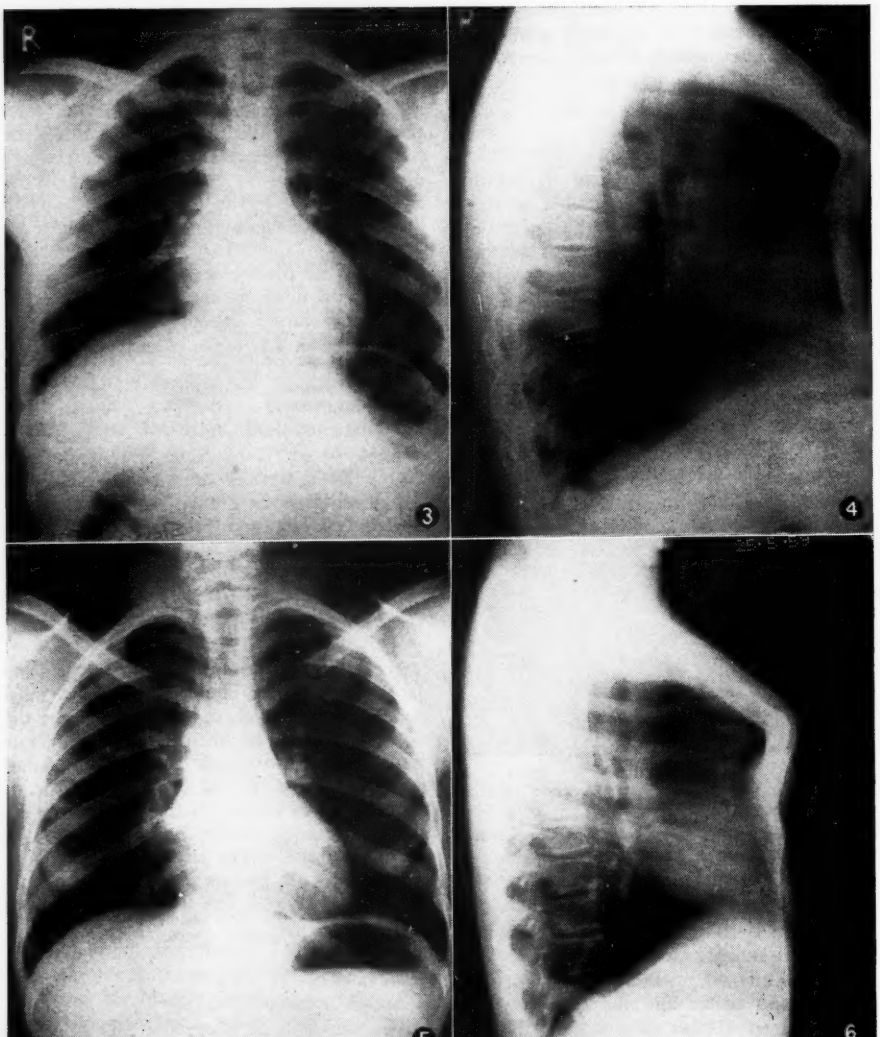

Fig. 3. Stephen. Antero-posterior view.
Fig. 4. Stephen. Lateral view.
Fig. 5. Jacobeth. Antero-posterior view.
Fig. 6. Jacobeth. Lateral view.

In this the *substernal ligament* and the *central tendon* of the diaphragm are important.[2] In the pigeon breast the diaphragm is relatively too small to accommodate the large chest. Its tension is increased and the dome is lowered, thereby restricting its normal respiratory excursion. This results in an ineffective non-productive cough.

CORRECTION OF DEFORMITY

Mechanical. In the pigeon breast Pritchard[4] describes an apparatus with lateral springs which apply antero-posterior pressure on the sternum and spine, suitably padded. This prevents expansion in this direction. The apparatus has to be worn night and day for many months or even years. A case is illustrated with excellent comestic result. The child was 6 when the remedial apparatus was applied and was worn for 3 years.

Surgical. Indications for surgical correction are persistent cough, retarded development, and restricted activity due to dyspnoea on exertion and especially if the patient is eager to have it done for aesthetic reasons. Surgery is based on principles of thoracoplasty.[3]

The optimum age for operation and the technique employed depends on the type and extent of the deformity. Every case must be studied on its merits.

In the case of Stephen and Jacobeth surgical interference is not justified as they are in good health and the deformity does not embarrass them clinically or psychologically.

The operation is by no means trifling.[5]

SUMMARY AND CONCLUSIONS

An identical arcuate type of pigeon-breast deformity in a brother and sister is described.

The clinical features combine those of funnel and pigeon breast.

Diagnosis is determined by the 'dominant factor', which is the arcuate sternum. This belongs to pigeon-breast deformities.

Funnel chest and pigeon breast have a common etiology and pathogenesis.

In the etiology heredity plays an important role. The father and paternal grandfather of the two children had similar deformities.

In the pathogenesis there are two factors:

(*a*) Overgrowth of the second and third pairs of ribs; and

(*b*) Abnormal size and shape of the diaphragm.

Psychological disturbances may be marked.

Treatment is discussed in brief.

The arcuate type of pigeon breast is an unusual anterior chest wall deformity which receives scant mention in medical literature and merits wider recognition.

I wish to thank Dr. I. Frack, Medical Superintendent of Baragwanath Hospital, for permission to submit this paper for publication.

I am also indebted to Dr. H. Clain, Senior Radiologist, for the X-ray films; to Dr. S. Sachs, of Meadowlands Polyclinic, where the children were seen; and to Mr. D. S. Smith, Technician, Department of Surgery, University of the Witwatersrand, for the photographs.

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ELECTROSURGERY IN GENERAL PRACTICE

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Electrosurgery developed from the therapeutic use of diathermy for the production of heat in the tissues, and it was based on the fact that when tissues are submitted to a diathermy current or an oscillating current, it generates heat in the tissues. This is different from the heat applied externally, and is due to the effects of the current meeting the resistance of the tissues.

Electrosurgery is merely a small offshoot of the many developments which have followed the introduction of diathermy in the practice of medicine.

After a discussion of the basic principles we will describe as briefly as possible therapeutic uses, the dangers and the contra-indications for the use of this current in general surgery.

Electrosurgery or surgical diathemy denotes the application of electricity to the destruction of new growths and diseased tissue, and for cutting through normal tissue with minimal bleeding. In the strict sense of the term, the

electrocautery (the employment of metal tips or wires heated by the lighting current) and electrolysis for the removal of superfluous hair and for destruction of tissue, would also come under the heading of electrosurgery. Modern usage has, however, restricted the term electrosurgery to the surgical application of high frequency electrical currents.

There are 3 methods of high frequency heating in use at present. The oldest method, known as long wave diathermy, employs oscillations of a frequency of about a million per second, with a wave length of 300 metres, and is applied through bare metal electrodes placed in direct contact with the skin or mucous membrane. The newer method of short wave diathermy employs oscillations from 10 to 100 million per second, with a wave length of from 30 to 3 metres, and is routinely applied through a spacing of air, glass or rubber, but may also be applied by direct metallic contact. In the newest method of diathermy, micro-wave diathermy, oscillations of some 3,000 million per second, with a wave length of 1 metre to 10 centimetres are focussed in a single beam of electro-magnetic energy from some distance to the region to be treated. Micro-wave diathermy is not being used very much nowadays.

To produce oscillations of sufficiently high frequency and power to heat the human tissues, two essential changes have to be effected in the commercial alternating current which serves as the source of all high frequency electricity.

1. The voltage must be increased. This is done by a step-up transformer; and
2. The frequency must be increased. This is done by an oscillating circuit. Such a circuit consists of a condenser, inductance and a spark-gap in the spark-gap apparatus or long wave diathermy, and of a condenser, inductance and oscillating tubes in the vacuum type of apparatus or short wave diathermy.

The three principal divisions of the short wave diathermy apparatus are:

1. The power supply.
2. The oscillator.
3. The output or patient's circuit.

The Patient's Circuit. This consists of inductance and capacity. Either or both may be variable in order to 'tune' this circuit, but usually the coil is fixed and the condenser is variable. The tuning device of most short wave diathermy machines is this variable condenser. It serves to bring the patient's circuit into resonance with the oscillator frequency, just as one resonates a radio set when one tunes in to a station.

The long wave diathermy apparatus produces a 'damped' intermittent high frequency current. In operating a spark-gap apparatus the supply current is turned on through the main switch, and the amount drawn is regulated by the choke coil. The high frequency oscillations are regulated by the spark-gap control. At its lowest point no oscillations occur, and there is no current flow in the patient's circuit.

The short wave diathermy apparatus produces an 'undamped' sustained high frequency current.

Either the spark-gap or the tube type of diathermy machine is used for surgical diathermy, and an apparatus is now available from which one may either get the spark-gap or tube type of diathermy, or a mixture of both the spark-gap and the tube type of diathermy.

The principle of action of all electrosurgical methods is an intense concentration under a small electrode.

If a homogeneous substance is subjected to the diathermy current by means of two equally sized pads placed on either side, theoretically there would be an equal distribution of heat between the two pads (Fig. 1A).

If now one uses a large pad and a small pad there is a concentration of the heat towards the smaller pad (Fig. 1B).

To carry this still further (and in practice this is done with electrosurgery) a large dispersal electrode is used while the active electrode is now a small cutting blade, pin-point or a ball-point (Fig. 1C). It will now be seen that the concentration is at the pin-point, and this permits of electrosurgery. It must be impressed and remembered that the dispersal electrode is also an 'active electrode' and a source of potential danger.

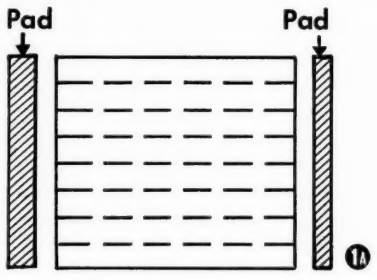
The result of this is a destructive heat effect which leads to instantaneous coagulation of the albumin of the tissues. The extent and form of tissue destruction varies with the type of current and the technique of application. Heated metals in the form of the electrocautery have been used for a long time for the destruction of superficial tissues and malignancies, but their action is superficial and the heat cannot be maintained at an even temperature. If they are too hot, normal, as well as malignant tissue, is destroyed. With the high frequency electrosurgical apparatus, the scalpel or needle remains cold all through the operation, the heat generated is in the tissues themselves, and its extent is always under the perfect control of the operator.

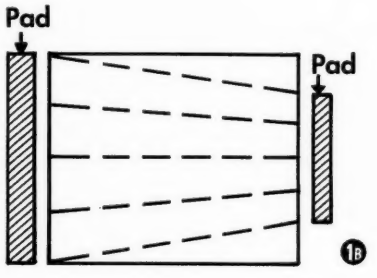
Electrosurgical methods have come to fill a very definite role in both major and minor surgery. When first introduced in the form of desiccation and coagulation, they were welcomed for their effectiveness in local tissue

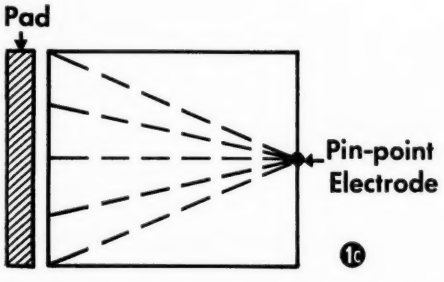
destruction. From the original field of tumour surgery the newer method of electrical cutting and its modification have brought electrosurgery into the field of general surgery, enabling the successful attack on vascular organs anywhere in the body and leading to a new surgical technique.

For purposes of description and technique, surgical diathermy is divided into:

1. Electrodesiccation.
2. Electrocoagulation.
3. Electrosection.







Electrodesiccation. A needle-point electrode is connected to the single high voltage (Oudin) terminal of the long wave diathermy apparatus and held in contact with or at a slight sparking distance, to the area to be treated. The current employed is one of comparatively high voltage and low amperage and its oscillations are damped. When this current is applied to the tissues at appropriate strength and duration, drying and shrinkage of the parts occur, due to the evaporation of the water content of the cells. A dry mass remains which can be curetted away or left in place to slough off in due time.

Histological changes in the cells after electrodesiccation show them to become shrunken and shrivelled with their nuclei condensed or elongated, with a suggestion of cell outline, the whole assuming a mummified appearance (mummification necrosis). The blood vessels become thrombosed with no evidence of haemorrhage.

Since the mode of cell death is associated with very little degenerative change and scant disintegrated material, there is but a small amount of fibrous tissues as an end result. This explains the good cosmetic results and the fact that the neighbouring healthy tissues are spared the devitalization resulting from the formation of abundant contractile fibrous tissue.

Electrocoagulation. The current employed in electrocoagulation is one of higher amperage and of damped oscillations. There exist two techniques in electrocoagulation.

1. The usual method is to connect an 'active' needle or other pointed electrode to one terminal of a high-frequency apparatus and a large dispersing one to the other terminal. Whenever the active electrode is brought into contact with the tissues, a grayish-white discolouration of varying depth appears, corresponding with widespread coagulation of these tissues.

2. A modified method of electrocoagulation consists in the employment of two active electrodes, two needle-points on a specially constructed clamp electrode. This is called biterminal coagulation. As a result of this technique, coagulation only occurs in the small area between the two active electrodes.

Following electrocoagulation one finds histologically that the cell outline is entirely lost, the effected tissue elements become fused into a structureless homogeneous mass presenting an appearance not unlike that of hyalinization. This greater degree of destruction results finally in a proportionally greater amount of fibrous (scar) tissue formation.

Electrosection. A needle-point or thin blade is connected to one terminal of an apparatus producing a current of undamped oscillations, and a large dispersive electrode is connected to the other terminal. With appropriate strength of current, the active or operating

electrode applied to the tissues will glide through them with ease. Like butter under a hot knife, the tissues almost melt under the influence of the cutting current.

The histological effects occur in two zones. The innermost area is a zone of molecular disruption due to the explosion of cells. There is no clear explanation of this process as yet. One explanation is that the assumption by the molecules in the tissues of a high rate of vibration causes dissolution of the molecular structure, another explanation is that the sudden expansion of the cell, due to intensive heating, converts its liquid contents into explosive steam.

In the outer zone there is evidence of elongation of tissue cells and a slight amount of coagulation. The width of these zones equals about 0.1 mm. when the average cutting strength is used.

The high frequency resistance of the various tissues accounts for a certain variation of resistance to electrical current. There is much more resistance when cutting through fat than through skin and muscle, hence more power must be used. (Skin, 289 ohms; voluntary muscle, 110 ohms; bone, 1800 ohms; fat, 2180 ohms).

Healing of Electrosurgical Wounds. It has been found that it is not possible to produce a purely cutting effect without some necrosis on a skin with a horny layer, but this slight necrosis does not always preclude healing by primary intention. In the mid-period of healing the electrical wounds are weaker, as shown in animal experiments by comparison of the tensile strength in the wounds.

Healing of wounds by the cutting current in skeletal muscles and the muscular wall of the stomach and intestines, has been found entirely comparable in strength to scalpel wounds and without secondary necrosis or infection.

Specific Effects on Blood Vessels. The most important argument in favour of electrosurgery is the effect on the three classes of blood vessels.

1. *Very Small Vessels, Capillaries, Arterioles and Venules.* These are vessels of a size which do not usually bleed after a clamp is removed during operation. When tissues containing such vessels are severed with a cutting current without any zone of coagulation, there is momentarily some capillary haemorrhage, but within a few minutes this haemorrhage ceases completely. In organs with oozing capillary beds, such as the liver, spleen, brain, kidney and in muscles, this result offers a revelation to most surgeons. Histological sections showed obstruction of the lumen by endothelial lining cells and wrinkling of the vessel wall. Laboratory investigations have demonstrated an abundant liberation of thrombokinase from the vascular wall. The latter phenomenon accounts for the sudden and effective haemostasis by the cutting current. A flash, a simple division of the tissue by the cutting current is sufficient to produce the sealing effect.

2. *Small or Moderate-sized Vessels, which Bleed after Taking the Clamp off and Must be Ligated in Ordinary Surgery.* These vessels are usually not obliterated by the cutting current but can be successfully sealed off by the coagulation current. The usual method of doing this is to touch either the haemostat which grips them, or touch the vessel directly by the tip of the coagulating electrode. The less tissue coagulated the less necrosis follows and the less the chance of secondary haemorrhage or infection from the thrombosed blood vessel.

3. *Larger Blood Vessels of the Size of the Radial Artery or Larger.* In these vessels the customary ligation is preferable to electrical haemostasis.

ADVANTAGES OF ELECTROSURGERY

On the basis of the changes described the advantages claimed for electrosurgery are:

1. Lessening or elimination of bleeding, the immediate sealing of blood vessels and lymph vessels gives not only a clearer and better operating field, but also sterilizes it to a great extent.

2. Facilitating the rapid and gentle handling of even the most delicate tissue.

3. Lessening of operative shock and after pain, through decrease of the capillary wound surface, through slowing up the resorption of toxic tissue products and by destruction of afferent nerve paths of the pain.

4. Promoting more rapid healing in the average case and leading to a softer and a less disfiguring scar.

APPARATUS FOR ELECTROSURGERY

Two types of high frequency apparatus serving for electrosurgery are:

1. The spark-gap type producing damped oscillations and used for heavy coagulation; and
2. The tube type producing undamped current used for electrosection and light coagulation.

The most modern electrosurgical unit produces a spark-gap type of current, and the tube type of current, as well as a mixture of the

spark-gap and the tube type currents. This so-called blended current is used where cutting and coagulating is required simultaneously. These machines are fitted with a triple foot switch so that the surgeon can select:

(*a*) A pure coagulating current;

(*b*) A pure cutting current;

(*c*) A mixture of coagulating and cutting so as to cut and coagulate at the same time.

Both circuits have separate controls enabling the operator to select suitable amounts of both currents and to blend according to the requirements when the mixed current is required.

TECHNIQUE OF ELECTROSURGICAL COAGULATION

For the active electrode in electrocoagulation, a needle or small sized ball attached to an insulated holder is used. A dispersive electrode is used in both electrocoagulation and electrosection in order to complete the electrical circuit through the body.

One must always bear in mind that there is basically no difference between the active and the dispersive electrodes. There is the same amount of current passing through both and because the active electrode is smaller there is a concentration of the current at the one spot, whereas the current is dispersed over a wider area of the skin through the dispersive electrode.

The dispersive electrode consists of a fairly large plate of diathermy metal, about 8″ by 10″ in size. This is best placed under the buttocks at a safe distance from the field of operation. There must be good contact between the metal and the skin at all times and it must be well insulated by sheet rubber from the operating table or other metal parts.

The dispersive electrode should be rolled perfectly flat before use each time, so as to avoid poor or uneven contact due to surface irregularities. Sharp points may cause burns.

It is advisable to securely fix the dispersive electrode by an elastic bandage so as to prevent its displacement if the patient moves.

The use of the term 'indifferent' or 'inactive plate' instead of dispersing plate should be avoided, because it misleads beginners to believe that no action takes place beneath this electrode and, therefore, no attention has been paid to it. As a matter of fact severe burns have occurred under improperly applied dispersive plates and the victim of such an occurrence will hardly call the means of such unpleasant effect an 'indifferent' one.

TECHNIQUE OF ELECTROSURGICAL CUTTING

A thin flat blade or needle serves as the active electrode and the usual large metal plate as the dispersing electrode. The thinner the edge of the cutting electrode, with the same current strength and speed of cutting, the less coagulation occurs along the line of separation. If primary union is desired, a thin needle or very thin blade should be used for skin incisions with a quick cutting action. Large needles are used for heavier coagulation and haemostasis such as incising vascular tumours, the lip or tongue or lung abscesses. Thin loops, oval and round, are used for taking biopsy specimens or piecemeal removal of other tissues.

Haemostasis. The superiority of electric cutting over the non-electric scalpel is at once evident in operations on the liver, lung, kidney, spleen, thyroid and brain, with their extensive capillary beds. The sealing of all small blood vessels gives a clearer and better operating field and a faster operating time. If larger blood vessels are severed and are not sealed by the cutting current, they should be grasped with the usual haemostatic forceps. At a convenient time during the operation, the clamp held in the operators' left hand, should be touched about 1″ or 2″ from its end by the active electrode—not with its tip, because the heavy sparking may damage the edge. Within one or two seconds a thin visible zone of coagulation appears around the tip of the forceps. The circuit should now be broken and the forceps removed. In using this technique, care should be taken to avoid grasping excess tissue about the vessel itself within the jaws of the artery clamp, to avoid excessive necrosis in the surrounding tissue.

Under-water Cutting. Under water cutting is a modification of electrical cutting which enables excision of tumours in the bladder and the prostate. It is done with the usual biterminal technique, but the active electrode must be insulated as far as the cutting tip to ensure minimum of current leakage through the conductive medium of water. There is also need of considerable current strength to make up for this inevitable leakage. The ordinary uninsulated electrosurgical knife or loop operating from a low power machine cannot act under water.

ANAESTHESIA IN ELECTROSURGERY

In conditions requiring general anaesthesia there is a danger of explosions occurring if Cyclopropane, ether, ethyl chloride or Vines-

thene are used in connection with electrosurgery. Even the smallest spark may cause a violent explosion.

AVOIDANCE OF BURNS IN ELECTROSURGERY

(*a*) *From the Dispersive Electrode.* Basically there is no difference between the active and the dispersive electrode. To prevent burns from this dispersive electrode the following precautions should be taken:

1. Always make sure that this electrode makes good contact with the skin over the whole surface.

2. Do not place the dispersive electrode over bony prominences as the current would tend to concentrate over the bony projections and give rise to burns.

3. Make sure that the electrode cannot move by bandaging the electrode to the place desired. It often happens that the patient either is moved or moves on the table and the good contact is broken producing burns.

4. If there is a large amount of hair on the skin to which the dispersive electrode is applied, then it is advisable to shave the hair off so as to ensure good contact of the electrode with the skin.

5. Make sure that the dispersive electrode is insulated from the operating table by means of rubber sheeting.

6. Do not place the dispersive electrode at the side of the body, against an arm or against a leg, as proper contact with the whole surface of the electrode cannot be made and burns may result.

(*b*) *From the Active Electrode.* Burns have been known to have occurred from the active electrode itself. As a rule this is due to the active electrode, when not in use, being placed inadvertently close to the patient. At the same time, by accident, the operator may put his foot on the switch and this electrode then becomes active. The operator should always take the precaution of placing this electrode, when not in use, at some distance from the patient.

NOTES AND NEWS : BERIGTE

Dr. M. N. Teubes, M.B., B.S. (London), F.R.C.S. (Ed.), has commenced practice as a Specialist Surgeon at 713 Harley Chambers, Jeppe Street, Johannesburg. (*Telephones*:—*Rooms*: 22-2665; *Residence*: 42-3900).

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Dr. H. Rifkin has joined Dr. M. H. Finlayson, Dr. H. W. Clegg, Dr. A. S. Peden and Dr. P. W. J. Bosman in practice as a Specialist Pathologist at 601 Dumbarton House, Church Street; and 305 Medical Centre, Heerengracht, Cape Town. (*Telephone:* 2-7521.)

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LAMINARIN SULPHATE FROM SEAWEED FOR ATHEROSCLEROSIS

The New Scientist (1959, **6**, 998) reports that seaweed yields the major constituent of a new drug now undergoing clinical trials in the treatment of atherosclerosis. The drug is made by reacting laminarin, a starch-like substance extracted from seaweed, with a sulphate. The product of this reaction, laminarin sulphate, exihibis marked anti-lipaemic activity; in other words, it reduces the fat content of the blood, so clearing the serum which tends to become turbid after meals.

Similar effects can be achieved with heparin, but this suffers from the disadvantage of prolonging the clotting time of blood and can therefore be administered for only limited periods. The new compound has only one twentieth of the anti-coagulating power of heparin, but more or less matches it in anti-lipaemic action.

Laminarin sulphate has been developed by the Boots Pure Drug Company, Ltd. in its Pharmacology Division at the new Biological Research Centre at Nottingham.

UNIVERSITY OF NATAL

FACULTY OF MEDICINE

NOVEMBER MEDICAL GRADUANDS—AWARD OF PRIZES

Association of Surgeons Prize: A. B. L. Pupuma
Burroughs Wellcome Prize: P. L. Patel
Arthur Goldsmith Prize: A. B. L. Pupuma
Prize in Medicine: C. T. Maitin
Prize in Paediatrics: C. C. C. Matondo
Horace Wells Medal: C. C. C. Matondo
Good Companions Award: S. P. N. Shongwe.

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DR. SEYMOUR HEYMANN

At the meeting of the Executive Board of the American Academy of Pediatrics held in October 1959, Dr. Seymour Heymann of Johannesburg was elected to Honorary Fellowship in the American Academy of Pediatrics.

Honorary Fellowship in the Academy is an honorary classification for distinguished physicians residing outside the Americas who have in some way made outstanding contributions to child health. The Executive Board conferred this honour upon Dr. Heymann in recognition of his many contributions to the health and welfare of children.

* * *

ANTI-HAEMOPHILIC GLOBULIN (BOVINE)

Westdene Products (Pty.) Ltd. announce that they now hold supplies of Anti-Haemophilic Globulin (Bovine) manufactured by Maws of England.

AHG is specifically for use as a prelude to surgery and in other emergencies involving cases of classical haemophilia. Reports, e.g. Lancet, 10 August 1957, pp. 251–259, have shown that in some instances AHG may be a life-saving measure. It is not intended to replace conventional methods such as the use of fresh human blood or plasma, and its use should be reserved for emergencies involving cases of classical haemophilia which cannot be dealt with by other means.

AHG is supplied in ampoules containing approximately 200 units and is administered by intravenous injection or drip. Dosage will depend upon the AHG level which obtains in the patient at the commencement of treatment. Treatment can be continued for a period of up to 10 days, provided no allergic reaction is noticed in this time. Should it become necessary to continue the treatment beyond this period, then it is necessary to change to AHG (Pig) supplies of which would have to be imported from England. The price to a hospital is 44s. 5d. for 100 units.

Caution: AHG, like other protein injections, can be potentially dangerous. In its present form the same type of AHG should not be given a second time to the same patient since this may cause a severe anaphylactic reaction. It should therefore only be used at present in those cases where life is in danger and not for any lesser incident.

Limited stocks of AHG Bovine (Maws) are being held by Westdene in Johannesburg and can be sent by air to most parts of the Union at a moment's notice. Small emergency supplies are available in Johannesburg, Cape Town and Durban at any time of the day or night at the addresses given below.

Further details regarding AHG may be obtained from Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg. *Telephone*: 23-0314.

Johannesburg: Day—Westdene Products (Pty.) Ltd., Feltex House, Commissioner Street, *Telephone*: 22-1761. Night—Mr. L. H. Reichenberg, *Telephone*: 42-1871; Mr. H. O. Evans, *Telephone*: 42-7154.

Cape Town: Day—Westdene Products (Pty.) Ltd., 408 Grand Parade Centre, *Telephone*: 41-1061. Night—Nite-Kem, Corporation & Darling Streets, *Telephone:* 2-3964.

Durban: Day—Westdene Products (Pty.) Ltd., 66–67 National Mutual Bldgs., *Telephone*: 2-4975. Night—Medicine Chest, 161 Berea Road, *Telephone*: 2-2221.

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BURROUGHS WELLCOME AND COMPANY OPEN FACTORY IN JOHANNESBURG

Prof. E. H. Cluver, Director of the South African Institute of Medical Research, officially opened Burroughs Wellcome and Company's factory at 130, Main Street, Johannesburg, on 1 December.

The factory has been built above the offices of the company in the central area of Johannesburg to facilitate the distribution throughout Southern Africa of the products of the Wellcome Foundation Limited —an organization which is without parallel in the pharmaceutical industry.

The Wellcome Foundation is registered as a Private Limited Liability Company, but all the shares are vested in five Trustees.

The Wellcome Trust, a recognized public charity, has for its main objects the advancement throughout the world of research in human and veterinary medicine and allied sciences, and the establishment or endowment of research museums and libraries.

Dr. Fred Wrigley, a director of the Wellcome Foundation, flew out from the United Kingdom to attend the opening of the Johannesburg factory.

This is the first time that ' B. W. & Co.' products are being manufactured and packaged in Africa. The equipment and design of the new establishment are based on the Wellcome Foundation's factory in the United Kingdom—one of the most modern of its kind in the world—and adapted to local conditions.

The colour scheme of the entire factory is designed to combine the maximum light and reflection with the minimum amount of strain on the workers. In the ampoule sorting section, for example, where the work is particularly tedious on the eyes, green predominates as being the most restful colour. And in the large packing room where the majority of workers are employed, a radiogram is installed. A spacious recreation room offers off-duty relaxation for the employees.

This latest Burroughs Wellcome contribution to the pharmaceutical industry in South Africa is, in fact, a model of efficiency, hygiene and enterprise.

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COLLEGE OF GENERAL PRACTITIONERS CAPE OF GOOD HOPE FACULTY

UPJOHN TRAVELLING LECTURES

The Upjohn Company has offered to finance a series of lectures to be given by teams of specialists to country practitioners, who are members of the Cape of Good Hope Faculty of the College of General Practitioners.

These lectures will take the form of week-end refresher courses in selected towns and villages in the country. The first lecture in every area will be an open lecture and a courtesy invitation will be extended to every practitioner in the district. The lectures will be arranged throughout each year and will be known as the *Upjohn Travelling Lectures*.

It is intended that the first lectures will take place early in the New Year and suggestions and requests regarding subjects and venues will be most welcome.

All those interested are invited to write to the Chairman of the Cape of Good Hope Faculty of the College of General Practitioners, Dr. F. E. Hofmeyr, 18 Worcester Road, Sea Point, Cape Province.

UPJOHN-REISVOORLESINGS

Die Upjohn Maatskappy het aangebied om 'n reeks voorlesings te finansier wat deur spanne spesialiste aan praktisyns op die platteland, wat lede is van die Fakulteit Kaap de Goede Hoop van die Kollege van Algemene Praktisyns, gegee sal word.

Hierdie lesings sal die vorm aanneem van naweekopknappingskursusse in dorpe en dorpies op die platteland wat spesiaal vir die doel uitgekies sal word. Die eerste lesing in elke gebied sal 'n ope lesing wees en welwillendheids-uitnodigings sal aan elke praktisyn in die distrik gestuur word. Die lesings sal jaarliks dwarsdeur die jaar gereël word en sal bekend staan as die *Upjohn-reisvoorlesings*.

Dit word in die vooruitsig gestel dat die eerste lesings vroeg in die nuwe jaar sal plaasvind en voorstelle en versoeke oor onderwerpe en plekke van byeenkoms sal graag ontvang word.

Almal wat belangstel word uitgenooi om te skryf aan die Voorsitter van die Fakulteit Kaap de Goede Hoop van die Kollege van Algemene Praktisyns, dr. F. E. Hofmeyr, Worcesterweg 18, Seepunt, Kaap.

PREPARATIONS AND APPLIANCES

PROTONAMINE

A CONCENTRATED ORAL AMINO ACID, VITAMIN, MINERAL SUPPLEMENT

Westdene Products (Pty.) Ltd. announce the introduction of *Protonamine*, a concentrated oral amino acid, vitamin, mineral supplement. This product has been developed following extensive study and research into nutriment requirements in South Africa by Transvaal Protone Products (Pty.), Ltd.

In *Protonamine* every effort has been made to incorporate all the components which are essential or beneficial to life. It will supply all 10 of the essential amino acids together with 8 of the dispensable amino acids. These are derived from proteins of both animal and vegetable origin. In addition to calcium, phosphorus and iron, 11 trace elements and 10 vitamins are provided in the correct proportions. Carbohydrate has been added in the optimum amount to ensure a high protein sparing action. The low fat content of *Protonamine* consists of unsaturated fats. Many additional factors such as a high saline content to ensure the solubility of the amino acids, have also been carefully taken into account to enhance the nutritional value of this product.

Protonamine is indicated for use in cases where metabolic deficiencies arise, such as chronic disease, nutritional and hypoproteinaemia, pregnancy and lactation, paediatrics, geriatrics, convalescence, pre- and post-surgical treatment, burns, etc. Particular attention has been paid to the palatability and ease of incorporation into the normal hospital diet.

Protonamine is supplied in a powder form in 1 lb. jars. One tablespoonful is added to hot water to prepare a pleasant hot beverage. It is suggested that this quantity should be given 3 times a day.

Further details may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

PERMITIL

A NEW ANTI-ANXIETY AGENT

Scherag (Pty.) Ltd. announces from White Laboratories, Inc. Kenilworth, N.J., U.S.A., the discovery of a new anti-anxiety agent, *Permitil*. Broadly described as a phenothiazine tranquillizer, it has a a wide spectrum at low milligramme dosage.

Chemistry: Chemically, *Permitil* is 1-(2-hydroxyethyl) - 4 -3 - (2 - trifluoromethyl - 10 - phenothiazinyl)-propyl-piperazine dihydrochloride.

Indications: *Permitil* rapidly relieves symptoms of anxiety, tension, agitations and emotional unrest, without hypnotic depressant effect, impaired alertness or slowed intellectual function. Psychomotor overactivity is lessened. *Permitil* enables the physicians to provide effective pharmacotherapy for many of the emotional problems seen in general practice. It is useful in the management of anxiety associated with organic diseases, those diseases where anxiety and tension play a contributing part, e.g. gastrointestinal tension states, neurodermatitis, asthma, premenstrual tension, arthritis, hypertension and tension headache.

Dosage: Must be adjusted to the individual, but generally one 0.25 mg. tablet twice a day, should suffice (*Permitil* has rapid onset of action and is long lasting).

It is seldom necessary to go beyond a total of 2 mg. per day, where there is insomnia, or where an hypnotic action is desired, other medication (e.g. barbiturates) may be added.

Side Effects and Precautions: An occasional feeling of jitteriness or restlessness should not be interpreted as increased anxiety requiring greater dosage. Usually this effect is temporary and disappears. Autonomic effects, such as dry mouth, blurred vision, constipation, etc. are rare, as are depressant effects such as drowsiness or dizziness.

Being a phenothiazine derivative, physicians should be alerted to the possibility of jaundice and agranulocytosis, although they have not been reported in studies up to 2 years. Likewise extra-pyramidal reactions such as Parkinsonism, dystonia, dyskinesia may occur, but seldom below 3 mg. a day and are readily controlled by anti-Parkinson drugs (*Artane*), sedation and withdrawal of *Permitil*. *Permitil* is contraindicated in comatose or depressed states, or those with a convulsive history, and its anti-emetic effect may obscure signs of toxicity due to other drugs, brain tumour or intestinal obstruction.

REFERENCES

Ayd, Frank J., Jr. *Fluphenazine: Its Spectrum of Therapeutic Application and Clinical Results in Psychiatric Patients*. Read at the American Psychiatric Association, Divisional Meeting, Seattle, Washington, 24-27 September 1959.

Darling, Harry F. *Fluphenazine: A Preliminary Study*. Dis. Nerv. System, **20**, 167, April 1959.

PARABAL

Westdene Products (Pty.) Ltd. announce the introduction of *Parabal*, the detoxicated form of phenobarbitone discovered in the research laboratories of the West Pharmaceutical Company Limited of England.

Parabal enables the dosage of phenobarbitone to be reduced by 80% without loss of sedation. This striking result is achieved by combining phenobarbitone with an amino acid derivative. Absorption of the new presentation is much more rapid than that of phenobarbitone itself and the excretion rate is far greater. As a result, the advantages of phenobarbitone therapy are maintained but the side effects such as 'hangover' and 'dopiness' are eliminated.

Parabal is indicated in the treatment of psychosomatic disorders, personality upsets characterized by marked anxiety or nervous tension, hypertension, simple insomnia, intensive and neurotic symptoms associated with the menopause, etc In addition, trials being carried out in Johannesburg indicate that *Parabal* has a definite place in epilepsy therapy.

The average dosage for *Parabal* is one tablet night and morning for adults and this may be increased if necessary to 2 tablets 3 times a day. *Parabal* may be administered with safety to children, the usual dosage being ¼ to ½ tablet, according to age. This low dosage emphasizes a further advantage of *Parabal*, which is its low cost compared with other sedative drugs.

Parabal is available in bottles of 100 and 500 tablets.

Further information may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

REVIEWS OF BOOKS

SURGICAL PATHOLOGY

Surgical Pathology. By Lauren V. Ackerman, M.D. (1959. Pp. 1079 + Index. With 1114 Figs. 127s. 6d.). St. Louis: C. V. Mosby Company.

According to the author, this book has been written for medical students, surgeons, pathologists, radiologists, etc. The medical student can derive some benefit by reading it, although he has to beware of dogmatic statements such as the assertion on p. 630 dealing with adenomyosis that, 'the endometrium and stroma reflect the changes of the menstrual cycle.' Novak has pointed out that the aberrant endometrium of endometriosis is often completely unresponsive to progesterone. The author's assertion that the breakdown of Hodgkin's disease into Hodgkin's paragranuloma, Hodgkin's granuloma and Hodgkin's sarcoma is not warranted, is certainly not above criticism. Jackson and Parker have pointed out the far better prognosis for patients with Hodgkin's paragranuloma. The photomicrograph on page 718 purporting to show an example of a malignant cystosarcoma phyllodes looks very benign, although this is one of the few poor photographs. The chapter dealing with *Bone and Joint Diseases* has been brought up to date and is probably one of the best sections in the book.

The book gives a fairly good outline of surgical pathology to the general surgeon and would be useful to him in preparation for post-graduate examinations.

The photographs and photomicrographs are excellent and maintain the high standard of most American textbooks. Probably the most useful aspect of this book to every member of the medical fraternity is the excellent list of references at the end of each chapter.

For this second edition of his book, Dr. Lauren V. Ackerman has enlisted the aid of a surgeon, Dr. Harvey R. Butcher, and there is as a result some improvement in the sections dealing with clinicopathological correlation. There are still, however, numerous platitudes and meaningless statements in this book. For example, in the *Introduction* on page 22:

'The surgeon we choose to operate has not only technical dexterity (a fairly common commodity) but, more important, good judgment and a personal concern for his patient's welfare.'

This and other similar passages, could well have been left out of the second edition. A similar, somewhat unnecessary, dissertation is the long paragraph on p. 727 on how to prepare a poor section of a lymph node. The impression is also given in many parts of the book, that the author is attempting to compress too much in a book of just over 1,000 pages, approximately half of which are illustrations. As a result, some subjects are dealt with woefully inadequately. For example, gonococcal salpingitis is summarily dismissed in 3 lines. Although gonorrhoea may have ceased to be a problem in the United States (and this is doubtful), it certainly is still a source of concern to the gynaecologist in many other countries, and chronic inflammation is not the only histological feature. In the section dealing with endometrium, cystic glandular hyperplasia is not even mentioned.

The chapter on *Wound Healing* has been enlarged and vastly improved and a section on the *Surgical Pathology of the Eyes and Ocular Adnexa* has been added. The latter chapter is a good one and the subject is dealt with fairly completely.

SYNOPSIS OF ANORECTAL DISEASES

Synopsis of Treatment of Anorectal Diseases. By Stuart T. Ross, M.D., F.A.C.S., F.I.C.S. (1959. Pp. 235 + Index. With 79 Figs. 55s. 3d.). St. Louis: C. V. Mosby Company.

Although there are many books devoted to anorectal disease, there is always room for others, especially if they add to knowledge of this important branch of surgery.

To-day, when medical students have a comprehensive curriculum to cover, many of them, on qualification, feel the need of a small pocket book on such a subject.

Ross' *Synopsis of Treatment of Anorectal Diseases* fills the need of the recently qualified doctor, and will help him in his approach to this difficult subject. More damage is wrought in this area than anywhere else by an inadequate knowledge of the anatomy and the disease.

On the subject of anorectal abscesses, the author stresses a most important factor in the treatment, viz. that conservative therapy is unavailing and nothing is to be gained by waiting for the abscess to point except a further period of misery and a larger eventual lesion. The time to open an abscess is when the diagnosis is made.

This synopsis should also be of great value to the general practitioner who performs his own surgery.

BONE AND JOINT TUBERCULOSIS

Surgical Treatment of Bone and Joint Tuberculosis. By Robert Roaf, M.A., M.Ch.Orth., F.R.C.S.Ed., F.R.C.S.Eng., W. H. Kirkaldy-Willis, M.A., M.D., B.Chir., F.R.C.S.Ed., and A. J. M. Cathro, M.B., Ch.B. (1959. Pp. 134 + Index. With 90 Figs. 30s. Postage 1s. 7d.). Edinburgh and London: E. & S. Livingstone Ltd.

This book is timely because of the tremendous changes that have taken place during the last decade in the incidence of tuberculosis and in its treatment.

In England and America, as a result of chemotherapy and improved control of tuberculosis, a number of surgical tuberculosis centres have been abolished or transformed into hospitals catering for other disabilities.

Unfortunately this salutary state of affairs has not yet descended upon Asia or Africa, and surgeons in these continents have now had thrust upon them the unsolicited reputation of being the worlds' experts on bone and joint tuberculosis. Since the population of these two continents is so vast and so rapidly increasing, it is of the utmost importance for all doctors associated with surgical tuberculosis to be aware of these modern trends in its treatment.

The authors of this short monograph (two of them are from Nairobi and one from Oswestry) have wisely condensed their subject and laid stress mainly on recent advances. They apologise for being dogmatic but need not have done so for

'dogma and diagram' contribute to systematic teaching.

The diagrams depicting anterolateral fusion of the spine, extraperitoneal, extrapleural and transpleural, are extremely helpful and the descriptions of these operations are concise and devoid of padding.

Only two favoured methods of arthrodesis of the hip are mentioned, viz. the intra-articular stapling arthrodesis and the abduction osteotomy arthrodesis.

In South Africa, where tuberculosis is also still rampant, a preference is shown for other types of arthrodesis operations of the hip, but this is of no great consequence for, as we say in Afrikaans, 'Elke man soen sy vrou op sy eie manier.' Translated surgically it indicates that, whatever the method employed, intra-articular arthrodesis of joints for tuberculosis has come to stay.

This reviewer enjoyed reading this book. He liked the *Foreword* by Dr. Harold Boyd and, despite the fact that he does not understand why the peroneus tendons must be carefully sutured in subtaloid arthrodesis (p. 99) and why no mention is made of streptohydrazid, he can warmly recommend it to all the surgical and orthopaedic tuberculosis experts in Asia and Africa and other parts of the world where the Mycobacterium still lurks menacingly.

CORRESPONDENCE

MANIPULATION FOR RELIEF OF PAIN

To the Editor: Allow me to congratulate Dr. Philip Dalgleish on his excellent article concerning *Pain and its Relief by Manipulation* (28 November 1959).

Being a confirmed manipulator myself, I can bear out every word he says. We do indeed get results, almost unbelievable at times, and we have the everlasting gratitude of patients.

I am so glad that he has given us some scientific grounds to answer our sceptical colleagues at medical meetings, etc., being, as they unfortunately still are, in the overwhelming majority.

J. G. Botha,
B.A., M.B., Ch.B.

'La Mer,'
Plettenberg Bay.

INTERNATIONAL CONGRESS OF GASTROENTEROLOGY

To the Editor: In the summer of this year we forwarded our first announcement on the International Congress of Gastroenterology, which will be held from 20 to 24 April 1960, at Leyden and Noordwijk aan Zee, the Netherlands.

The two main themes of the Congress will be:

(*a*) Pathology, pathological physiology and clinical aspects of the small intestine;

(*b*) Hepatitis, cirrhosis and their possible connexion.

The detailed provisional programme is available on request and may be consulted for any further information about the Congress, both scientific and practical.

May we emphasize the fact that our Congress is not only open to gastroenterologists, but also to all those who are interested in gastroenterology by profession, physicians as well as scientists.

C. Schreuder, M.D.,
General Secretary.

16, Lange Voorhout, The Hague,
The Netherlands.

IMMUNIZATION PROCEDURES

To the Editor: As a general practitioner I have been following with interest the articles and correspondence on *Immunization Procedures.*

The latest letter from Dr. Pincus Catzel in the issue of 31 October 1959 raises a very important point, viz. the cutting down of the number of injections.

However, I find that I cannot agree with his suggested programme of immunization. In his letter he suggests including the polio vaccine with the DPT vaccine, starting at 4 months. Whereas such an idea would be of benefit, I cannot help feeling that it would instil a false sense of security, the reason being that adequate response cannot be achieved before the age of 8 months. (*See* Prof. Wilfrid Gaisford *in* The Practitioner, September 1959, page 281; and Dr. H. J. Parish, *Ibid.*, page 277).

Dr. Parish states that '7 months is the earliest age at which uniformly good response to active immunization can be expected'. My own practice has been as follows:

3 months—Smallpox vaccination.
4 months—DPT.
5 months—DPT.
6 months—DPT.
7 months—Polio.
8 months—Polio.
15 months—3rd Polio.
Pre-Nursery School (± *4 years*)—4th polio and DPT booster.

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CANCER CURRENT LITERATURE INDEX

To the Editor: We have pleasure in announcing a new Excerpta Medica publication in the field of *Cancer.*

We are continuing to publish the *Cancer Abstracts Section* (*XVI*), but we have introduced a Priority Abstracts System whereby a number of important medical journals in the field of cancer are abstracted without delay by members of our Editorial Staff in Amsterdam. The introduction of this policy means that readers of *Cancer Abstracts* will have access to the most important literature published in this wide field with the minimum of delay.

The new publication which Excerpta Medica is now privileged to announce is entitled *Cancer Current Literature Index.* This important new service is being published by the Excerpta Medica Foundation for the American Cancer Society, New York.

This new title service will enable cancer investigators everywhere to have an 'advance guide' to all the world's cancer literature.

Excerpta Medica.

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The Netherlands.